COMPARISON OF SALIVARY GLUCOSE AND SERUM GLUCOSE CONCENTRATION IN NON-INSULIN DEPENDENT DIABETES MELLITUS PATIENTS - A CASE CONTROL STUDY

Dissertation submitted to

THE TAMILNADU Dr. M.G.R.MEDICAL UNIVERSITY

In partial fulfillment for the Degree of

MASTER OF DENTAL SURGERY

BRANCH IX

ORAL MEDICINE AND RADIOLOGY

MARCH 2012

CERTIFICATE

This is to certify that this dissertation titled **"Comparison of Salivary Glucose and Serum Glucose concentration in Non-Insulin Dependent Diabetes Mellitus patients"** is a bonafide record of work done by **Dr. Ruchi Gera** under my guidance during her postgraduate study period **2009-2012.**

This dissertation is submitted to **THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY,** in partial fulfillment for the degree of **MASTER OF DENTAL SURGERY, BRANCH IX – Oral Medicine and Radiology.**

It has not been submitted (partial or full) for the award of any other degree or diploma.

Guided By:

2/11/11

RAGAS DENTAL COLLEGE & HOSPITAL CHENNAI

Dr. S. Shanmugam, M.D.S,
Professor and Head
Department of Oral Medicine & Radiology
Ragas Dental College & Hospital
Chennai – 600 119.

Dr. S. SHANMUGAM, M.D.S.,
Prof. & H.O.D. Oral Medicine & Radiology
RAGAS DENTAL COLLEGE & HOSPITAL
2/102, East Coast Road Uthandi
Chennai - 600 119.

2/11/11

Dr. S. Ramachandran, M.D.S,
Principal,
Ragas Dental College & Hospital
Chennai – 600 119.

PRINCIPAL
RAGAS DENTAL COLLEGE AND HOSPITAL
UTHANDI, CHENNAI 600 119

Date: 02.11.2011

Place: Chennai - 600 119

ACKNOWLEDGEMENTS

Words seem less to express my deep sense of gratitude to my post graduate teacher, mentor, guide, ***Dr. S. Shanmugam, M.D.S.****, Professor and Head of Department, Department of Oral Medicine and Radiology, Ragas Dental College and Hospital, for his valuable guidance, tireless pursuit for perfection, constant support and encouragement throughout my post graduate curriculum. I can never thank him enough for his perseverance throughout my study period. I also thank him for all his kind help without which this dissertation would not have been possible.*

I take this opportunity to thank ***Dr.S.Ramachandran, M.D.S.,*** *Principal, Ragas Dental College and Hospital, for his generous support rendered throughout the course.*

I would like to thank my Professor ***Dr.Capt.S.Elangovan, M.D.S.,*** *for his valuable guidance.*

I am extremely thankful to ***Dr.S.Kailasam, M.D.S.,*** *and* ***Dr.Capt.S.Manoj Kumar, M.D.S.,*** *for their constant support and encouragement.*

I am extremely indebted to ***Dr. P.E Chandra Mouli****,* ***Dr. L.Arvind, Dr. Ramalakshmi and Dr.M.Shuba*** *for their encouragement and support rendered throughout the course.*

I am truly indebted to ***Dr. B. Anand*** *who always took out time to help me and guide me through various aspects of my post graduate studies.*

I am extremely thankful to ***Dr.P.Mahesh Kumar*** *who showed an immense amount of patience in explaining the statistical analysis time and again.*

I earnestly thank ***Mrs. Kalyani*** *and* ***Mr. Balaji*** *of Department of Biochemistry, V.H.S, Adyar hospital for all their help.*

A sincere appreciation is expressed to ***Mr. Ravanan****, Statistician for helping me with the statistical analysis.*

I would like to thank ***Mr.K.Thavamani*** *and* ***Ms.R.Sudha*** *for the immense help with the printing and binding of this dissertation.*

I also express my profound sense of gratitude to all the subjects who participated in the study without whom the study would not have been possible.

I thank my parents, my sister and my brother in law for being the most wonderful, encouraging and understanding family and showering their constant love and blessings.

I would like to thank all my batch mates ***Dr. S. Srividhya****,* ***Dr. Malavika, Dr. Ramasubramaniyan*** *and* ***Dr. B. Senthil*** *as well as other post graduate colleagues for their immense support.*

It is very rightly said that "Friends are the family that we choose". I would like to take this opportunity to thank ***Dr. Savitha, Dr. Ashish, Mr. Tushar*** *and* ***Dr. Sahai*** *for being such wonderful friends and most importantly for being there always.*

*I would also like to thank **Dr. Christeffi Mabel** for being the most wonderful senior and for teaching me all that she did and **Dr. Lisa** for always being so enthusiastic and willing to help out.*

*Above all I thank **The Lord Almighty** for without whose grace nothing would have been possible.*

LIST OF ABBREVIATIONS

S.NO	ABBREVIATION	EXPANSION
1.	DM	Diabetes Mellitus
2.	IDDM	Insulin-Dependent Diabetes Mellitus
3.	NIDDM	Noninsulin-Dependent Diabetes Mellitus
4.	DM1	Type 1 Diabetes Mellitus
5.	DM2	Type 2 Diabetes Mellitus
6.	FPG	Fasting Plasma Glucose
7.	OGTT	Oral Glucose Tolerance Test
8.	ADA	American Diabetic Association
9.	IFG	Impaired Fasting Glucose
10.	IGT	Impaired Glucose Tolerance
11.	MHC	Major Histocompatability Complex
12.	GCF	Gingival Crevicular Fluid
13.	DAN	Diabetic Autonomic Neuropathy
14.	PG	Parotid Gland
15.	SSG	Submandibular Salivary Gland
16.	SLG	Sub Lingual Gland
17.	AUC	Area Under the Curve Over Baseline
18.	AMC	Age Matched Control
19.	SOD	Superoxide Dismutase
20.	Agah	Active Gherlin
21.	dGAH	Inactive Gherlin
22.	QOL	Quality Of Life

23.	HbG	Glycosylated Hemoglobin
24.	AIDS	Auto Immune Deficiency Syndrome
25.	CG	Capillary Glucose
26.	ER	Excretion Ratios
27.	SGH	Salivary Gland Hypofunction
28.	SS	Sjogren Syndrome
29.	RNFPG	Random Non Fasting Plasma Glucose
30.	FDR	First Degree Relatives
31.	SOD	Superoxide Dismutase
32.	HbA1c	Hemoglobin A1c
33.	PGL	Plasma Glucose level
34.	SGL	Salivary Glucose level
35.	SpH	Salivary pH
36.	TBARS	Thiobarbituric Acid Reactive substance
37.	MDA	Malondialdehyde
38.	AOA	Total antioxidant activity
39.	LEADER	Leicester Ethnic Atherosclerosis and Diabetes Risk
40.	ND	Non-Diabetic
41.	DC	Diabetic Children
42.	Na^+	Sodium Ion
43.	Cl^-	Chlorine Ion
44.	K^+	Potassium Ion
45.	HCO_3^-	Bicarbonate Ion
46.	BMI	Basal Metabolic Index

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

CONTENTS

DIABETES MELLITUS (DM) is a complex multisystemic metabolic disorder characterized by a relative or absolute insufficiency of insulin secretion and/or concomitant resistance to metabolic action of insulin on target tissues.[1] The two predominant forms of DM are known as Type I or Insulin Dependent Diabetes Mellitus (IDDM) and Type II or Non–Insulin Dependent Diabetes Mellitus (NIDDM) [2]. NIDDM accounts for more than 90% of the diagnosed cases of DM[2]. Globally 140 million people are estimated to have DM. It is estimated that there will be over 230 million people with DM by the year 2010, and half of this population will be in Asia[3]. The number of people with diabetes in India currently around 40.9 million is expected to rise to 69.9 million by 2025 unless urgent preventive steps are taken[4]. This metabolic disease is a burden on both patients and society because of the high morbidity and mortality associated with infections and renal, retinal and vascular complications. Primary prevention of the disease and the prevention of diabetic complications are of great practical importance[5].

SALIVA is a complex fluid, whose important role is to maintain the well being of the oral cavity[6]. 1000 to 1500 ml of saliva is secreted per day and is approximately about 1ml/min. Parotid gland (PG) contributes to about 25% of the saliva while Submandibular (SSG) 70% and Sublingual (SLG) 5% respectively. Mixed saliva from all the glands is slightly acidic with a pH of 6.35-6.85 and is hypotonic to plamsa. Mixed saliva contains about 99% water and 1% solids. The remaining 1% consists of most part of

the large organic molecules inclusive of proteins, glycoproteins, lipids, small organic molecules like glucose, urea and electrolytes[6]. Saliva plays many important roles in the oral cavity. It helps in preparation of food for swallowing, helps in appreciation of taste, plays a role in digestion, has cleansing and protective functions, plays an important role in speech, and regulates the body temperature and the water balance. Parotid glands produce a watery secretion. Submandibular gland and sublingual gland produces more viscous fluid than parotid gland[7]. The importance of well functioning salivary glands for oral health is well known.

The composition and secretion of saliva is influenced by local as well as systemic, hormonal, nutritional and metabolic factors.[7] Diabetes Mellitus which is known to alter the constiution and flow of saliva[8]. Although differences in the output and composition of saliva from Diabetic and Non Diabetic subjects have been observed in a number of studies, many of these findings have been contradictory.

Saliva offers some distinctive advantage. Whole saliva can be collected non-invasively and by individuals with limited training. However, studies pertaining to the use of saliva as a non invasive tool in monitoring blood glucose levels in Diabetic patients have been done predominantly in the Western population.[9,10]

This study is an attempt to Estimate and Correlate the Salivary Glucose concentration and Serum Glucose concentration in Diabetics and healthy controls in a Chennai Population.

AIM:

Comparison of Salivary Glucose and Serum Glucose concentration in Non-Insulin Dependent Diabetes Mellitus patients.

OBJECTIVES:

1. To estimate the Salivary Glucose and Serum Glucose concentration in Non-Insulin Dependent Diabeties Mellitus patients.
2. To estimate the Salivary Glucose and Serum Glucose concentration in Healthy control group.
3. To correlate these Salivary Glucose and Serum Glucose concentrations in Non Insulin Dependent Diabeties Mellitus patients and Healthy controls.

A review of Diabetes Mellitus and Saliva are being presented here.

DIABETES MELLITUS

Diabetes Mellitus (DM) is a disease of glucose, fat, and protein metabolism resulting from impaired insulin secretion, varying degrees of insulin resistance, or both[11]. Hyperglycemia is the most clinically important metabolic aberration in DM and the basis for its diagnosis. Apart from the obvious impact of impaired glucose metabolism, DM and chronic hyperglycemia are associated with important ophthalmic, renal, cardiovascular, cerebrovascular, and peripheral neurological disorders[12].

CLASSIFICATION OF DIABETES MELLITUS

Most cases of DM can be classified as Type 1, formerly, known as Insulin-Dependent Diabetes Mellitus (IDDM) and Type 2 formerly, known as Noninsulin-Dependent Diabetes Mellitus (NIDDM). Blood glucose elevation that does not satisfy the definition of Type-1 or Type-2 DM is classified as impaired glucose tolerance or impaired fasting glucose. Secondary forms of DM also exist. For example, diseases of the pancreas, such as pancreatitis, may produce a state of absolute insulin deficiency. Numerous drugs may create a Diabetic state, glucocorticoids being the most notable. Glucocorticoids not only increase insulin resistance in liver and muscle, but also impair the response of pancreatic beta cells to elevated plasma glucose[12]. Recognition of secondary forms of DM is important because removal or management of the underlying cause can reverse the Diabetic condition. Another form of DM is Gestational Diabetes or DM

presenting during pregnancy which is the result of insulin production insufficient to overcome insulin resistance produced by placental anti-insulin hormones like estrogen, prolactin or cortisol[12].

EPIDEMIOLOGY OF DIABETES IN INDIA

Mohan et al[4] reviewed the epidemiology of Type 2 Diabetes in the Indian scenario. The authors explained that India leads the world with largest number of Diabetic subjects earning the dubious distinction of being termed the "Diabetes capital of the world". According to the **Diabetes Atlas**[13] published by the International Diabetes Federation, the number of people with Diabetes in India currently around **40.9** million is expected to rise to **69.9** million by 2025 unless urgent preventive steps are taken. The so called "Asian Indian Phenotype" refers to certain unique clinical and biochemical abnormalities in Indians which include increased insulin resistance, greater abdominal adiposity i.e., higher waist circumference despite lower body mass index, lower adiponectin and higher high sensitive C-reactive protein levels[4]. This phenotype makes Asian Indians more prone to Diabetes and premature coronary artery disease[4]. The most disturbing trend is the shift in age of onset of Diabetes to a younger age in the recent years. Early identification of at-risk individuals would greatly help in preventing or postponing the onset of Diabetes and thus reducing the burden on the community and the nation as a whole[4].

REGULATION OF BLOOD GLUCOSE

Diabetes has an impact on a number of fundamental metabolic processes. Glucose homeostasis is the result of the relative influences of two opposing hormones, insulin and glucagon[14]. Insulin is a protein synthesized in the pancreatic beta cells. It exerts its biochemical effects by interacting with transmembrane cellular receptors. The principal role of insulin is to facilitate storage of glucose as glycogen, free fatty acids as triglycerides, and amino acids as protein[15]. Insulin also inhibits the breakdown of glycogen, lipids, and protein. Furthermore, insulin inhibits ketogenesis and gluconeogenesis. Insulin therefore has its most important effect on muscle and adipose tissues and on the liver. Glucagon supports opposing activity by stimulating glucose and fatty acid formation, ketogenesis, and conversion of amino acids to glucose[12]. Following a meal, plasma insulin increases, altering the relative activity of insulin and glucagon in favor of insulin. As a result, dietary carbohydrate is stored in muscle and liver in the form of glycogen. Free fatty acids are converted to triglycerides in fat and amino acids are converted to protein. As plasma glucose returns to its preprandial value, so too does insulin secretion, and the preprandial insulin/glucagon ratio is reestablished. The sensitivity of target tissue to insulin is an important determinant of insulin effect. Feedback mechanisms increase insulin release in individuals who are relatively insulin resistant and decrease insulin release if there is increasedtissue sensitivity[12]. Target-tissue

insulin sensitivity plays an important role in the pathophysiology of Type-2 DM.

ETIOLOGY

In both the common types of DM, enviromental factors interact with genetic susceptibility to determine which people develop the clinical syndrome and the timing of its onset. However, the underlying genes, precipitating enviromental factors and pathophysiology differ substantially between Type 1 and Type 2. Type 1 is invariably associated with profound insulin deficiency requiring replacement therapy. Type 2 retains the capacity to secrete some insulin but exhibit impaired sensitivity to insulin and can usually be treated without insulin repalcement therapy. However, upto 20% of patients with Type 2 Diabetes ultimately develop profound insulin deficiency requiring replacement therapy.[11]

PATHOPHYSIOLOGY OF TYPE-1 DIABETES

Rossini AA et al[16] characterized Type-1 DM by an absolute insulin deficiency brought about by the Autoimmune destruction or accelerated disappearance of pancreatic beta cells. However, some patients have no evidence of an Autoimmune mechanism. **Libman IM et al**[17]described such patients to have Type-1B DM. **Martin S et al**[18]described mononuclear lymphocytic infiltrates, principally T lymphocytes and **Eisenbarth et al**[19] identified them in pancreatic islets in individuals with Type-1 DM. **Littorin B et al**[20] identified autoantibodies to a number of beta-cell antigens can be in the sera of those with Type-1 DM. Such autoantibodies can be detected

well in advance of the onset of clinical Diabetes and in some first-degree relatives of individuals with Type-1 DM. **Riley WJ et al**[21] realised that, high autoantibody titers in relatives of Diabetics are harbingers of the development of Clinical Diabetes within a few years. **Feutren Get al**[22] said that novel Immunosuppressive treatment of recently diagnosed Type-1 DM can decrease or even eliminate the need for exogenous insulin administration.

However, the potential toxicity of continuous immunosuppressive therapy precludes its clinical application in DM treatment. Susceptibility to Type-1 DM is inherited and the principle gene associated with this genetic predisposition is the Major Histocompatability Complex (MHC) on chromosome 6. **Davies JL et al**[23] found out that a number of HLA genes have been implicated in the familial clustering of Type-1 DM. **Atkinson MA et al**[24] said that a life-long risk of developing Diabetes is 6% in offspring and 5% in siblings of affected individuals. Apart from the underlying role of genetics, environmental factors are also believed to play an important role in the pathogenesis of Type-1 DM. **Dahlquist GG et al**[25] said that several pregnancy and perinatal factors, such as maternal age >25 years, preeclampsia, neonatal respiratory disease, and jaundice, have been associated with the development of Type-1 DM. **Szopa TM et al**[26] study revealed that viral infection has also been implicated in the destruction of beta cells or as a trigger for the production of autoantibodies. **Genuth SM et al**[27] found out that the pancreas has a substantial reserve for insulin

production and clinical DM does not occur until 90% of beta cells have been eliminated. The end result of an absolute insulin deficiency is impaired glucose uptake by muscle and fat as well as a loss of insulin-induced suppression of liver glucose production. **Genuth SM et al**[28] found out that FPG may rise to 300 to 400mg/dl and Post-prandial levels as high as 500 to 600mg/dl. This produces an osmotic diuresis with polyuria and, subsequently, increased thirst. Plasma fatty acid levels increase as does hepatic uptake of free fatty acids. This in turn, leads to increased production of ketoacids and metabolic acidosis which is known as Diabetic Ketoacidosis[12]. Weight loss occurs as a result of protein catabolism and lypolysis.

PATHOPHYSIOLOGY OF TYPE-2 DIABETES

Kahn CR et al[29] said that the pathophysiology of Type-2 DM is complicated by the fact that patients present with varying degrees of both insulin deficiency and insulin resistance. **Boden G et al**[30] said that in contrast to Type-1 DM, hyperglycemia in Type-2 DM is principally a result of insulin resistance. **Cavaghn MK et al**[31] said that the eventual loss of the ability of the pancreas to increase insulin output, in the setting of insulin resistance, creates a relative insulin deficiency and progression to established Type-2 DM. Hyperglycemia itself may contribute to insulin deficiency through a toxic effect on pancreatic beta cells. The practical implication of this complex interaction between insulin resistance and insulin production is that any clinical measure taken to normalize plasma

glucose will improve glucose homeostasis. Although adverse effects on fatty acid metabolism are seen, in contrast to Type-1 DM, there is usually sufficient residual insulin secretion in Type-2 DM to limit ketoacid formation and prevent the development of clinical acidosis. **Groop LC et al**[32] said that some Type-2 Diabetics also manifest pancreatic islet-cell autoantibodies typical of Type-1 DM and experience a more rapid decline in beta-cell function than those without autoantibodies. In contrast to Type-1 DM, genetics significantly influence the development of Type-2 DM. **Benett PH et al**[33] found out that the lifetime risk for a first-degree relative of an affected individual is 5 to 10-fold the risk in an age and weight-matched population without a family history of DM. **Mokdad AH et al**[34] said that obesity, especially of long duration, is an important risk factor for the development of Type-2 DM. **Chan JM et al**[35] said that abdominal obesity (waist >102 cm in men, >88 cm in women), in particular, is an important risk factor for Type-2 DM and is associated with insulin resistance. Type-2 DM is often accompanied by other conditions in addition to obesity. These include hypertension, elevated serum low-density–lipoprotein cholesterol, low serum high-density–lipoprotein cholesterol. **DeFronzo RA et al**[36] said that the clustering of metabolic risk factors for both Type-2 DM and Cardiovascular disease has prompted the diagnosis of ‘‘Metabolic Syndrome’’.**Eckel RH et al**[37] said that the Metabolic Syndrome is considered a pro-inflammatory, prothrombotic state that is a significant predictor of Type-2 DM and Cardiovascular Disease.

CLINICAL FEATURES

Diabetes can affect almost every system in the body. Examination of the patient with Diabetes is focused on hands, blood pressure, eyes, insulin injection sites and feet[11]. Examination of the hands may show limited joint mobility. There is presence of painless stiffness in the hands and it occassionally affects the wrists and shoulders. Dupuytren's contracture is common in Diabetes and may include nodules or thickening of the skin and knuckle pads[11]. Carpal tunnel syndrome is common in diabetics and presents with wrist pain radiating into the hands. A trigger finger may also be present at times. Muscle wasting or sensory changes may be present as features of a peripheral sensorimotor neuropathy, though more commonly seen in the lower limbs[11]. Eyes show impaired visual acuity and cataract or lens opacification eventually[11]. Insulin injection sites show bruising, lumps, subcutaneous fat deposition and erythema. Look for evidence of callus formation on weight bearing areas, clawing of the toes, loss of the plantar arch, discoloration of the skin, localised infection and presence of ulcers[11]. Fungal infection may affect skin between toes and nails. There is usually weight loss in IDDM and obesity in NIDDM.

ORAL MANIFESTATIONS OF DIABETES MELLITUS

GrossiS et al[38], Tsai C et al[39], Taylor GW et al[40] and Karjalainen KM et al[41] said that independent of the severity of plaque accumulation there will be presence of gingivitis with gingival bleeding, periodontitis, and periodontal bone loss with DM, especially when poorly controlled. Marked

mobility of teeth and generelised attrition is an important hallmark of Diabetes. Defects in immune status, altered bacterial flora, and microvascular disease are the postulated pathogenesis of Diabetic periodontal disease[42]. **Iacopino et al**[43] said that evidence also indicates that bacteremia associated with periodontitis contributes to insulin resistance and destruction of pancreatic islet cells. Diabetic patients may complain of dry mouth. Xerostomia may be a manifestation of hyperglycemia-associated dehydration or impaired salivary gland function[44]. Oral candida infections occur with greater frequency in poorly controlled Diabetics.[45]

INVESTIGATIONS

Testing urine for glucose is a common procedure for detecting Diabetes, using sensitive glucose specific dipsticks[11]. Tesing should be performed on urine passed 1-2 hours after a meal since this will detect more cases of Diabetes than a fasting specimen. Glycosuria always warrants a further assessment by blood testing. The greatest disadvantage of using urinary glucose as a diagnostic screening procedure is the indiviual variation in renal threshold for glucose[11]. The most common cause of glycosuria is a low reanl threshold which is common in young people and during pregnancy[11]. Estimation of the blood glucose concentration, using an accurate laboratory method rather than a side-room technique is therefore essential in making the diagnosis.

Ketone bodies can be indentified by the nitroprusside reaction, which is primarily specific for acetoacetate[11]. The test is conveniently carried out using tablets or dipsticks for ketones. Ketonuria may be found in people fasting or exercising strenuously for long periods, who have been vomitting repeatedly, or who have been eating a diet high in fat and low in carbohydrate[11]. Ketonuria is therefore not pathognomic of diabetes but if associated with glycosuria, the diagnosis of Diabetes is highly likely. In Diabetic Ketoacidosis, ketones can be detected in plasma using dipsticks.

Dipstick testing for albumin is a standard procedure to identify the presence of renal disease in people with Diabetes. This will detect urinary albumin greater than 300mg/dl[11]. Smaller amounts of urinary albumin can be measured and these provide indictors of the risk for developing Diabetic nephropathy and/or macrovascular disease.

Laboratory glucose testing in blood relies upon enzymatic reaction and is cheap, usually automated and highly reliable. However, variation in blood glucose depends on whether the patient has eaten recently[11]. Blood glucose can be measured with colorimetric or other testing sticks, which are often read with a portable electronic meter. These finger pricks are used for capillary testing to monitor Diabetes treatment.

Glucose concentration are lower in venous than in arterial or capillary blood. Whole blood glucose concentrations are lower plasma concentrations because red blood cells contain relatively little glucose[11]. In general, venous plasma values are the most reliable for diagnostic purposes.

Glycalated hemoglobin provides an accurate and objective measure of glycemic control over a period of weeks to months[11]. This can be utilized as an assessment of glycaemic control in a patient with known Diabetes, but is not sufficiently sensitive to make a diagnosis of Diabetes and is usually within the normal range in patients with Impaired Glucose Tolerance.

In Diabetes, the slow non-enzymatic covalent attachment of glucose to haemoglobin (glycation) increases the amount in the HbA_1 or HbA_{1c} fraction relative to non – glycated adult hemoglobin (HbA_0)[11]. This fraction can be separated by chromatography, laboratories may report glycated hemoglobin as total glycated hemoglobin (GHb), HbA_{1c}. The rate of formation of HbA_{1c} is directly proportional to the ambient blood glucose concentration, a rise of 1% in HbA_{1c}corresponds to an approximate average increase of 2mmol/l in blood glucose[11]. Although HbA_{1c} concentration reflects the integrated blood glucose control over the lifespan of the erythrocyte that is 120 days, half of the erythrocytes are replaced in 60 days and HbA_{1c} is weighted by changes in glycemic control occuring in the month before measurement[11]. As HbA_{1c} is affected more by recent than by earlier events, a large shift in blood glucose control is rapidly accompanied by a change in HbA_{1c}, detectable with in 2-3 weeks.

Various assay methods can be used to measure HbA_{1c}, precluding direct comparison of HbA_{1c} values between laboratories. HbA_{1c} estimates may be erroneously diminished in anemia or during pregnancy and may be difficult to interpret with some assay methods in patients who have anemia

or a hemoglobinopathy[11]. HbA_{1c} is usually measured once or twice a year to assess glycaemic control, permitting appropriate changes in treatment and identifying inconsistency with the patient's record of home blood glucose monitoring[11]. HbA_{1c} also provides an index of risk of developing Diabetic complications.

Glycated serum protein can be measured and because of their shorter half life, give an indication of glyacemic control over the preceeding 2 weeks.

The concentration of serum lipids – total choloestrol, low density and high density lipoprotein (LDL and HDL) cholestrol and triglyceride, is yet another important index of overall metabolic control in Diabetic patients and should be measured at diagnosis and regularly thereafter[11]. Ideally, the triglyceride concentration should be measured in the fasting state.

DIAGNOSIS

Symptoms of hyperglycemia include thirst or dry mouth, polyuria, polydypsia, polyphagia, nocturia, tiredness, fatigue, recent changes in weight, blurring of vision, pruritus vulvae, balanits (genital candidiasis), nausea, headache, prediliction for sweet foods, mood change, irritability, difficulty in concentrating and apathy[11].

When Diabetes is suspected, the diagnosis may be confirmed by a random blood sugar concentration greater than 11.0mmol/l or 199mg/dl. When random blood glucose values are elevated but are not diagnostic of Diabetes, glucose tolerance is usually assessed by either fasting

bloodglucose estimation or by the oral glucose tolerance test (OGTT)[11]. In oral glucose the patients is asked to follow an unrestricted carbohydrate diet for three days before the test. The patient should be fasting overnight atleast for 8 hours. The patient is asked to rest for 30 mins before the test, with no smoking and should be seated for duration of test. The plasma glucose is measured before and 2 hours after 75 g of glucose load.

The diagnostic criteria for Diabetes Mellitus recommended by **World Health Organization** suggests that[11]

A. If a patient complains of symptoms suggesting diabetes
    a. Test urine for glucose and ketones
    b. Measure random or fasting blood glucose. Diagnosis confirmed by
        i. Fasting plasma glucose ≥ 126 mg/dl
        ii. Random plasma glucose ≥ 200 mg/dl.

B. Indications for oral glucose tolerance test in a Diabetic
        i. Fasting plasma glucose 110-126 mg/dl
        ii. Random plasma glucose 140-199mg/dl

HbA_{1c} is not used for diagnosis.

COMPLICATIONS OF DIABETES

Clark CM et al[46] found out that the chronic elevation of plasma glucose leads to increased intracellular accumulation of glucose and its metabolic products. **Nathan DM et al**[47] found out that resulting long-term complications include microvascular disease of the eye namely retinopathy

and nephropathy and a variety of neuropathies. Diabetic retinopathy occurs in all forms of DM with the earliest manifestations being retinal microaneurysms. With progression, affected vessels become occluded and retinal infarctions follow. Vessel proliferation can lead to vitreous hemorrhage, fibroproliferative changes with retinal traction, and vision loss. Diabetic nephropathy affects 30% of patients with Type-1 DM and 4% to 20% with Type-2 DM[48]. Beginning as thickening of the capillary basement membrane, deposition of protein ultimately leads to glomerulosclerosis, impaired renal function, and progression to renal failure. If a person does not develop nephropathy after having Diabetes for 25 to 30 years, then it is unlikely he or she will develop the condition[49]. This is unlike Diabetic retinopathy, where risk continuously increases over time. Diabetic neuropathy has many possible manifestations[49]. The most common presentation is symmetrical altered sensation in the toes and feet. A minority of patients experience a painful, burning character to the neuropathy. Motor-nerve involvement is less common but may involve both cranial and peripheral nerves. Cranial nerve neuropathies may present with extraocular muscle weakness and double vision.

Finally, involvement of the autonomic nervous system can affect gastric motility, erectile function, bladder function, cardiac function, and vascular tone. Cardiovascular disease occurs with greater frequency in Diabetics than in the general population. 75% of Type-2 Diabetics die of cardiovascular disease[50]. As noted, Type-2 Diabetics with the metabolic

syndrome have a clustering of risk factors for cardiovascular disease. The prevalence of coronary artery disease in Type-2 DM with the metabolic syndrome is twice that in individuals without Diabetes or Metabolic Syndrome[51]. Coronary artery disease develops at an earlier age in Diabetics, and atypical anginal symptoms and congestive heart failure are a more common presentation[52].

Haffner SM et al[51]discovered that the risk of a first myocardial infarction in patients with DM is equal to that of recurrent infarction in nondiabetics. Though some disagree, it is generally held that Diabetes with poor plasma glucose control is associated with an increased risk of infection. Neutrophil adherence, chemotaxis, phagocytosis and bactericidal activity, and cell-mediated immunity are all compromised in the hyperglycemic diabetic[53,54]. The plasma glucose threshold for such granulocyte dysfunction is in the range of 198 to 270mg/dL[55]. Both granulocyte and T-cell dysfunction are reversed by the administration of insulin[56,57]. The practical implication of Diabetic-associated immune dysfunction is that optimal control of plasma glucose is important both in the prevention of infection and in the management of established infection.

SALIVA

The most commonly used laboratory diagnostic procedures involve the analyses of the cellular and chemical constituents of blood[7]. Other biologic fluids are utilized for the diagnosis of disease, and saliva offers some distinctive advantages. Whole saliva can be collected non-invasively,

and by individuals with limited training. No special equipment is needed for collection of the fluid. Diagnosis of disease via the analysis of saliva is potentially valuable for children and older adults, since collection of the fluid is associated with fewer compliance problems as compared with the collection of blood[7]. Further, analysis of saliva may provide a cost-effective approach for the screening of large populations.

DIAGNOSTIC APPLICATION OF SALIVA

Saliva can be considered as gland-specific saliva and whole saliva. Gland-specific saliva can be collected directly from individual salivary glands: parotid, submandibular, sublingual, and minor salivary glands[7]. **Navazesh et al**[58] realised that secretions from both the submandibular and sublingual salivary glands enter the oral cavity through Wharton's duct, and thus the separate collection of saliva from each of these two glands is difficult. The collection and evaluation of the secretions from the individual salivary glands are primarily useful for the detection of gland-specific pathology, i.e., infection and obstruction. However, whole saliva is most frequently studied when salivary analysis is used for the evaluation of systemic disorders.

Whole saliva or mixed saliva is a mixture of oral fluids and includes secretions from both the major and minor salivary glands, in addition to several constituents of non-salivary origin, such as gingival crevicular fluid (GCF), expectorated bronchial and nasal secretions, serum and blood

derivatives from oral wounds, bacteria and bacterial products, viruses and fungi, desquamated epithelial cells, other cellular components, and food debris[59].

Saliva can be collected with or without stimulation. Stimulated saliva is collected by masticatory action i.e., from a subject chewing on paraffin or by gustatory stimulation, i.e. application of citric acid on the subject's tongue[60]. Stimulation obviously affects the quantity of saliva; however, the concentrations of some constituents and the pH of the fluid are also affected. Unstimulated saliva is collected without exogenous gustatory, masticatory, or mechanical stimulation. Unstimulated salivary flow rate is most affected by the degree of hydration, but also by olfactory stimulation, exposure to light, body positioning, and seasonal and diurnal factors[7]. The best two ways to collect whole saliva are the draining method, in which saliva is allowed to drip off the lower lip, and the spitting method, in which the subject expectorates saliva into a test tube[61]. Saliva has protective properties and contains a variety of antimicrobial constituents and growth factors[62]. In addition, saliva has lubricating functions and aids in the digestion of food[60].

The salivary glands are composed of specialized epithelial cells, and their structure can be divided into two specific regions: the acinar and ductal regions. The acinar region is where fluid is generated and most of the protein synthesis and secretion takes place[7]. Amino acids enter the acinar cells by means of active transport, and after intracellular protein synthesis,

the majority of proteins are stored in storage granules that are released in response to secretory stimulation[63]. Three models have been described for acinar fluid secretion. These three models include the active transport of anions into the lumen and passage of water according to the osmotic gradient from the interstitial fluid into the salivary lumen. The initial fluid is isotonic in nature and is derived from the local vasculature. While acinar cells are water-permeable, ductal cells are not. However, ductal cells actively absorb most of the Na^+ and Cl^- ions from the primary salivary secretion and secrete small amounts of K^+ and HCO_3^- and some proteins. The primary salivary secretion is thus modified, and the final salivary secretion as it enters the oral cavity is hypotonic[64]. The autonomic nervous system that is the sympathetic and parasympathetic controls the salivary secretion. The signaling mechanism involves the binding of neurotransmitter primarily acetylcholine and norepinephrine to plasma membrane receptors and signal transduction via guanine nucleotide-binding regulatory proteins (G-proteins) and activation of intracellular calcium signaling mechanisms[64].

There are several ways by which serum constituents that are not part of the normal salivary constituents like drugs and hormones can reach saliva. Within the salivary glands, transfer mechanisms include intracellular and extracellular routes. The most common intracellular route is passive diffusion, although active transport has also been reported. Ultrafiltration, which occurs through the tight junctions between the cells, is the most

common extracellular route[65,66]. In contrast, a serum molecule reaching saliva by diffusion must cross five barriers: the capillary wall, interstitial space, basal cell membrane of the acinus cell or duct cell, cytoplasm of the acinus or duct cell, and the luminal cell membrane[66]. Serum constituents are also found in whole saliva as a result of GCF outflow. Depending on the degree of inflammation in the gingiva, GCF is either a serum transudate or, more commonly, an inflammatory exudate that contains serum constituents.

Some systemic diseases affect salivary glands directly or indirectly, and may influence the quantity of saliva that is produced, as well as the composition of the fluid. These characteristic changes may contribute to the diagnosis and early detection of these diseases.

Saliva can be analyzed as part of the evaluation of endocrine function. Insulin can be detected in saliva, and salivary insulin levels have been evaluated as a means of monitoring serum insulin levels. A positive correlation between saliva and serum insulin levels following a glucose tolerance test was reported for healthy subjects, Non-Insulin-Dependent Diabetic patients, and obese Non-Diabetic patients[67]. Similarly another study found a better correlation between salivary and serum insulin levels in 93 healthy subjects[68]. As assessed by radioimmunoassay, a glucose tolerance test performed on nine healthy patients produced a positive correlation between salivary and serum insulin levels. Salivary insulin levels reached maximal values approximately 30 minutes after the serum levels; 90 minvs. 60min[69]. Other investigators also reported a similarly high

correlation between salivary and serum insulin levels in healthy individuals and Insulin-Dependent Diabetic patients, but proposed that the use of salivary insulin levels for the evaluation of serum insulin levels could be misleading, since significant discrepancies between salivary and serum insulin levels were detected for several individuals[70]. Additional studies are required to determine if salivary insulin levels should be used for the evaluation of serum insulin levels.

In general, serum and salivary levels of protein hormones are not well-correlated. These hormones are too large to reach saliva by means of passive diffusion across cells or by ultrafiltration, and the detection of these hormones in saliva is primarily due to contamination from serum through GCF or oral wounds.

Salivary monitoring has many advantages over the more conventional serum analysis. Multiple saliva samples can be collected in a relatively short time interval, which makes the non-invasive collection of saliva ideal for this purpose.[71] These factors have to be considered when saliva is evaluated as an alternative for the evaluation of serum levels.

For accurate diagnosis, a defined relationship is required between the concentration of the biomarker in serum and the concentration in saliva. Normal salivary gland function is usually required for the detection of salivary molecules with diagnostic value. Salivary composition can be influenced by the method of collection and the degree of stimulation of salivary flow. Changes in salivary flow rate may affect the concentration of

salivary markers and also their availability due to changes in salivary pH. Variability in salivary flow rate is expected between individuals and in the same individual under various conditions. In addition, many serum markers can reach whole saliva in an unpredictable way. These parameters will affect the diagnostic usefulness of many salivary constituents[72].

Furthermore, certain systemic disorders, may affect salivary gland function and consequently the quantity and composition of saliva. Whole saliva also contains proteolytic enzymes derived from the host and from oral micro-organisms[73]. These enzymes can affect the stability of certain diagnostic markers. Some molecules are also degraded during intracellular diffusion into saliva. Any condition or medication that affects the availability or concentration of a diagnostic marker in saliva may adversely affect the diagnostic usefulness of that marker. Despite these limitations, the use of saliva for diagnostic purposes is increasing in popularity. Several diagnostic tests are commercially available and are currently used by patients, researchers, and clinicians.

Due to its many potential advantages, salivary diagnosis provides an attractive alternative to a noninvasive, time consuming, complicated, and expensive diagnostic approaches[7]. However, before a salivary diagnostic test can replace a more conventional one, the diagnostic value of a new salivary test has to be compared with accepted diagnostic methods. The usefulness of a new test has to be determined in terms of sensitivity, specificity, correlation with established disease diagnostic criteria, and

reproducibility. It is difficult to interpret the significance of a single report that examines levels of any particular marker[7]. However, due to the many potential limitations of salivary diagnosis, promising results from pilot studies must be confirmed in larger, well-controlled trials. While many questions remain, the potential advantages of salivary analysis for the diagnosis of systemic disease suggest that further studies are warranted. Definition of specific disorders that can be identified or monitored by the analysis of saliva offers the possibility of improved patient management.

Consequently, an increased utilization of saliva as a diagnostic fluid could be seen.

Sreedevi et al[71] estimated and correlated the salivary and serum glucose concentration in Diabetics and healthy controls. They included 60 newly diagnosed Type 2 Diabetic patients and 60 age and sex matched control subjects in their study. Blood and saliva samples from both the groups were collected at least two hours after breakfast. For the experimental group the samples were collected once again after the control of Diabetes. A highly significant correlation was found between salivary glucose and serum glucose before the treatment and also after the control of Diabetes. The correlation between the salivary glucose and serum glucose was also highly significant in the control group. The levels of salivary glucose did not vary with age and sex. In control group the salivary glucose ranged from 0.7 to 1.3% and the mean was 1.0 ± 0.1mg%. In the study group, before the treatment of Diabetes the salivary glucose ranged from 1.5

to 8.0mg% and the mean was 3.10 ± 1.04mg%. After the control of Diabetes, the salivary glucose ranged from 0.6 to 1.8mg% and the mean was 1.1 ± 0.2mg%. The comparison of salivary glucose before treatment and after control of Diabetes was done by using paired t-test as same samples were examined twice and difference was statistically highly significant that is P<0.001. Unpaired t-test was used to compare the Diabetic and control groups and the difference was statistically significant that is P<0.01. In control group the serum glucose ranged from 61 to 167mg% and the mean was 105.7 ± 22.3mg%. In study group, before the treatment of Diabetes, the serum glucose ranged from 205 to 490mg% and the mean was 309.5 ± 68.2mg%. After the control of Diabetes the serum glucose ranged from 71 to 167mg% and the mean was 119.7 ± 27.5mg%. The comparisons of serum glucose before treatment and after control of Diabetes was done by using paired t-test as same samples were examined twice and the difference was statistically highly significant that is P <0.01. The coefficient correlation r value for salivary and serum glucose in controls was +0.74. The value was found to be statistically highly significant that is P<0.001. The correlation coefficient value for salivary and serum glucose before the treatment of Diabetes was +0.67 and r value for salivary and serum glucose after Diabetes is bought under control was +0.66. The values were found to be statistically highly significant before the treatment of Diabetes and also after the control of diabetes wit a P<0.001. Salivary and serum glucose was correlated before the treatment of Diabetes and after the control of Diabetes

and also in control group in different age groups. It was observed that there was no significant correlation between different age and sex groups and salivary and serum glucose that is $p<0.05$. Hence, the authors concluded that there was a significant correlation between serum glucose and salivary glucose; salivary glucose holds the potential of being a marker in Diabetes. With a further added advantage of being a non invasive preocedure with no need of special equipments and with fewer compliance problems as compared with collection of blood.

Sashikumar et al[72] evaluated a total of 150 individuals between 40 and 60 years of age. 100 Diabetes Mellitus Type 2 were recruited and another 50 individuals without Diabetes were recruited. The subjects were divided into 3 groups. Group I consisted of 50 individuals with controlled Diabetes determined by random non fasting plasma glucose (RNFPG) values between 120mg/dl and 200mg/dl. Group II included 50 subjects with uncontrolled Diabetes determined by RNFPG values above 200 mg/dl. Group III was the control group without Diabetes with RNFPG 80 to 120mg/dl and was age and gender-matched with groups I and II. Subjects were recruited when presenting for routine follow-up, at which time blood samples were obtained for measuring glucose and glycosylated hemoglobin levels. One week later, subjects returned for delivery of a whole saliva sample and a second sample of blood.Such whole salivary samples represent fluids contributed by secretions from major and minor salivary glands and potentially, gingival crevicular fluid. Unstimulatedsalivary glucose (USSG)

levels were significantlyhigher in both uncontrolled and controlled Diabetes compared with Non Diabetes. Both SSG and USSG levels were significantly correlatedwith RNFPG in the entire study population. Only among those with uncontrolled Diabetes were SSG and USSG levels significantly correlated with RNFPG. It was concluded that although the concept of using salivary instead of blood glucose is intriguing, it does not seem to be biologically feasible, as the association between salivary and plasma glucose levels is not unambiguously established.

Hegde et al[73] performed a study to explore the potential of saliva as a diagnostic tool in which 26 Type 2 Diabetes patients were compared with 21 age matched Non-Diabetic healthy controls for Fasting plasma glucose (FPG) and salivary glucose (SG). Significantly high FPG with a $p = 0.005$ was found. FPG showed positive correlation to SG with $r = 0.410$ only in diabetes. Since SG levels did not differ between the two groups. Overall salivary glucose concentration showed no significant difference between two groups implying association of high plasma glucose with high SG levels to be an infrequent observation which may be affected by metabolic control of the disease. Significant positive correlation of FPG with SG in Diabetics further supports this aspect. It was concluded that conventional marker like FPG is a better indicator of glycemic status.

Veena et al[74] undertook a study in an attempt to compare and correlate glucose levels in saliva and serum of patients with Diabetes and

Non-Diabetic healthy individuals, to determine the efficacy of saliva as a diagnostic aid. They screened 250 individuals visiting Diabetic clinics randomly. Of these, 200 were confirmed Type 2 Diabetics and were under medication. The remaining 50 gave neither a past history of Diabetes nor did their present glycemic status depicted high values. Venous blood and salivary samples were obtained from each individual and subjected to glucose estimation. Both fasting and post-prandial samples were analyzed. In the study, glucose was detected in the saliva of both Diabetic and Non-Diabetics. The fasting salivary glucose values in the control group ranged from 4.1 to 13.3mg/dl and the postprandial salivary glucose values from 12.5 to 20.0mg/dl. The fasting salivary glucose values in the study group ranged from 4.1 to 26.6mg/dl and the Post-prandial salivary glucose values from 15.3 to 30.7mg/dl. It was observed that as blood glucose levels changed in both fasting and post-prandial samples, so did salivary glucose levels, irrespective of age and sex. A significant p value of <0.001 and positive correlation was found between blood glucose and salivary glucose levels in both the Diabetics and the controls. The authors concluded that saliva can be used as an adjunct diagnostic tool in Diabetes Mellitus.

S. SathyaPriya et al[75] studied a total of 60 patients, comprising 60 Type 2 Diabetic patients and 25 healthy controls for estimation of glucose in saliva, in order to aid in reaching firm conclusions about their alterations in Diabetics as compared to helathy Non Diabetics and to compare and correlate these parameters in Uncontrolled and Controlled Diabetics.

Salivary investigations were performed using unstimulated whole Saliva. A significant correlation was found between salivary and blood concentrations in the Diabetes.Mean salivary glucose levels were found to be significantly elevated in uncontrolled Diabetics when compared to healthy non-Diabetics. There was significant increase in mean salivary amylase, protein & potassium in Diabetic patients when compared to healthy Non-Diabetics. Furthermore, in this study the protein profiles of whole saliva of Diabetic and healthy Non-Diabetic were compared. The saliva from Diabetic patients appeared to have more of proline-rich protein bands. These findings suggested that saliva can be used reliably for reflecting and monitoring the blood glucose concentration in the patients of Diabetes Mellitus.

Cedric et al[76] evaluated salivary glucose concentration and excretion in unstimulated saliva in both normal and Type 2 Diabetic subjects. The authors found that in normal subjects, a decrease in saliva glucose concentration. The glucose concentration averaged 79.4 ± 5.8μM in unstimulated saliva in normal subjects. The glucose concentration averaged 187.3±20.0μM in unstimulated saliva of the Diabetics. The glucose concentration failed to differ significantly in male and female Diabetic patients. The glucose concentration in unstimulated saliva was about twice higher in the Diabetic patients (187.3 ± 20.0μM) than in the control subjects (79.4 ± 5.8μM). In the latter patients, as compared to control subjects, the relative magnitude of the increase in saliva glucose concentration was comparable, however, to that of blood glucose concentration. These findings

confirm the poor link between glycaemia and glucose concentration in saliva, atleast on an individual basis.

CampbellM.J.A. et al[77] in their study used two methods to analyse the saliva of non-diabetic and Type 2 Diabetic patients for glucose content. Using the Somogyi blood glucose estimation technique, glucose was detected only in the saliva of Diabetic patients. Blood glucose estimations were conducted on the same patients and no degree of correlation between blood glucose and salivary glucose could be demonstrated. Using Chromatographic techniques, spot tests, and an ultramicro-technique, glucose was found to be present in the saliva of both the Non-Diabetic and the Diabetic patient. At the same time other sugars were detected in the saliva of both groups of patients, namely galacturonic acid, glucuronic acid, lactose, maltose, sucrose, fructose, mannose, sorbose and arabinose, and a relationship between the coincident presence or absence of these sugars was calculated. A quantitative analysis of the glucose content showed that in those cases which reacted positively the glucose values for the Non-Diabetic lay between 0.24 and 3.33mg/100ml and for the Diabetic between 0.44 and 6.33mg/100ml.

Darwazeh et al[78] estimated the glucose concentration in unstimulated mixed saliva and serum was assayed and correlated with oral candidal colonization in 41 Type 2 Diabetic and 34 healthy control subjects. A statistically significant result was found, in Diabetic patients and it was

found that the salivary glucose concentration was significantly higher than in the controls and was directly related to blood glucose concentration.

Michael W. J. Dodds et al[79] studied whether improvements in the level of Diabetic control in a group of subjects with poorly controlled Non-Insulin-Dependent Diabetes Mellitus influence salivary composition. Repeated whole unstimulated saliva was collected from Diabetic patients attending an outpatient Diabetes education program and a matched Non Diabetic control group. Saliva was analyzed for composition. Subjects reporting taste alterations had higher mean blood glucose levels than subjects with normal taste sensation. They concluded that poorly controlled Non Insulin Dependent Diabetes Mellitus has no influence on saliva output, although amylase activity may be elevated, and there may be taste alterations

Amer et al[80] estimated the salivary and blood glucose concentrations in Non Diabetic healthy individuals and patients with Type 2 Diabetes Mellitus. Glucose could not be detected in the salivary samples obtained from the Non Diabetic control subjects whose random serum glucose concentrations were significantly higher ($p<0.005$) than the serum glucose concentrations of the Non Diabetic control subjects. Glycosylated haemoglobin A1c was also determined in the patients and a significant correlation with $r = 0.82$ was found between HbAlc and serum glucose concentrations in these patients, indicating that these patients had average elevated blood glucose concentration over anextended time period. Glucose

was only found in the saliva of patients with Diabetes Mellitus, while the salivary samples of age matched non-diabetic subjectsdid not show the presence of glucose. A significant correlation of r = 0.78 was found between salivary and blood concentrations in the Diabetics. This finding suggests that saliva can be used reliably for reflecting and monitoring the blood glucoseconcentration in the patients of Diabetes Mellitus.

Suleyman Aydin et al[81] examined the relationship between active (aGAH) and inactive (dGAH) ghrelin in the saliva and other salivary parameters in Type II Diabetic patients and healthy controls. Salivary parameters were assessed in a single measurement of unstimulated whole saliva from 20 obese and 20 non-obese Type II Diabetes patients, and in 22 healthy controls. Saliva aGAH and dGAH levels were measured using a commercial radioimmunoassay kit. Salivary concentrations of aGAH and dGAH ghrelin were more markedly decreased in obese Diabetic subjects than in the two other groups. Salivary glucose (200%) levels were significantly higher in obese Diabetic subjects than in controls ($p < .005$); and salivary glucose (192%) levels in Non-obese Diabetic subjects were also significantly higher than those ofcontrol. Salivary glucose levels in the obese Diabetic subjects were almost the same. Glucose levels were higher in diabetic subjects than in controls. Furthermore, there were correlations between GAH levels and BMI, and between GAH and blood pressure. These results indicate that saliva can be used as a valuable diagnostic aid in the relationship to other salivary parameters.

Meurman et al[82] investigated and studied the organic constituents of whole saliva in relation to autonomic nervous function in patients with 45 patients with mean age, 68± 6 years Non-Insulin-Dependent Diabetes and *77* control subjects (mean age, 67 ± years).. Resting whole saliva samples were collected and analyzed. There were no statistically significant differences between patients with Diabetes and control subjects in the organic constituents of saliva. They concluded that saliva secretion might be more affected by autonomic nervous dysfunction in patients with Non-Insulin-Dependent Diabetes than in Non Diabetic control subjects

Nakamoto et al[83] examined blood and saliva samples to see if there is a correlation between saliva glycated protein and blood glycated protein. Blood and saliva samples of 51 male workers were collected. They were divided into groups as control, and Diabetics. The fructosamine andhydrazine methods were used to measure saliva glycated protein. HbA1c, fructosamine and blood glucose were measured as indices of blood glycated protein, and the correlation between blood glycated protein and saliva glycated protein was examined. It was found that the saliva fructosamine glycated protein showed a significant correlation with HbA1c and blood glucose with a $r = 0.449$; $p = 0.001$ and $r = 0.445$; $p = 0.001$, respectively. No correlation was identified between saliva hydrazine glycated protein and the index of blood glycated protein. It was concluded that the blood glycated protein and blood glucose could be estimated by measuring saliva glycated protein.

Eliaz Kaufman et al[7] reviewed the diagnostic applications of saliva. In the review they examined the diagnostic application of saliva for systemic diseases. As a diagnostic fluid, saliva offers distinctive advantages over serum because it can be collected non-invasively by individuals with modest training. Furthermore, saliva may provide a cost-effective approach for the screening of large populations. Gland-specific saliva can be used for diagnosis of pathology specific to one of the major salivary glands. Whole saliva, however, is most frequently used for diagnosis of systemic diseases, since it is readily collected and contains serum constituents. These constituents are derived from the local vasculature of the salivary glands and also reach the oral cavity by the flow of gingival fluid. Analysis of saliva may be useful for the diagnosis of hereditary disorders, Autoimmune diseases, Malignant and Infectious diseases, and Endocrine disorders, as well as in the assessment of therapeutic levels of drugs and the monitoring of illicit drug use.

Maria-Sueli-Marques Soares et al[84] investigated and studied the concentration of salivary glucose in healthy individuals and compared it with the capillary glycemia. Samples of unstimulated whole saliva were collected from 63 Non-Diabetic patients. The concentration of salivary glucose and capillary blood was measured in all of the patients. The salivary glucose was determined by enzymatic method and spectrophotometry. The data was then analyzed and they found that the whole sample consisted of 47.6% males and 52.4% women, with an average age of 37.5±15.7yrs old.

The average blood glucose among the males studied was 100.05±13.51 mg/dl, and among females, it was 99.5± 13.9mg/dl. The average salivary glucose for the whole sample was 5.97± 1.87mg/dl, with 5.91± 2.19mg/dl among males and 5.97± 1.56mg/dl among females, respectively without any significant differences with p = 0.908.The concentration of salivary glucose did not present any statistically significant correlation with the capillary glycemia with p = 0.732. They concluded that the concentration of salivary glucose is not dependent on capillary glycemia and that the concentration of salivary glucose does not present significant differences between the measurements for males and females.

Bennett CM et al[85] assessd the validity of glycated haemoglobin A1c (HbA1c) as a screening tool for early detection of Type 2 Diabetes. They performed a systematic review of primary cross-sectional studies of the accuracy of HbA1c for the detection of Type 2 Diabetes using the oral glucose tolerance test as the reference standard and fasting plasma glucose as a comparison. They found 9 studies met the inclusion criteria. At certain cut-off points, HbA1c had slightly lower sensitivity than fasting plasma glucose (FPG) in detecting Diabetes, but slightly higher specificity. For FPG at a cut-off point of ≥ 6.1mmol/l, the sensitivity ranged from 48 to 64% and specificity from 94 to 98%. Both HbA1c and FPG have low sensitivity for the detection of impaired glucose tolerance. It was concluded that HbA1c and FPG are equally effective screening tools for the detection of Type 2

Diabetes. The HbA1c cut-off point of >6.1% was the recommended optimum cut-off point for HbA1c in most reviewed studies; however, there is an argument for population-specific cut-off points as optimum cut-offs vary by ethnic group, age, gender and population prevalence of Diabetes. It was concluded that the current cost of HbA1c is higher than FPG, the additional benefits in predicting costly preventable clinical complications may make this a cost-effective choice.

Mostafa S.A. et al[86] examined the potential impact of the preferred use of HbA1c as a diagnostic tool on the prevalence and phenotype of T2DM. They analysed the Leicester Ethnic Atherosclerosis and Diabetes Risk (LEADER) cohort for previously undiagnosed individuals between 40 and 75 years of age who had OGTT repeated if within the Diabetes range, and HbA1c results. They then compared the prevalence and phenotype of subjects with T2DM based on either HbA1c ±6.5% or OGTT using 1999 World Health Organization criteria. It was found that from the total population of 8696, they detected 291 (3.3%) with T2DM from using an OGTT, and 502 (5.8%) had HbA1c ≥6.5%. Of those diagnosed with T2DM by OGTT, 93 (1.2%) had HbA1c <6.5% and therefore would not have been classified as having T2DM using proposed criteria. Using HbA1c criteria resulted in 304 (3.5%) additional cases of T2DM, approximately doubling the prevalence. Of these 304 additional people, 172 (56.7%) had impaired glucose tolerance/impaired fasting glycaemia according to 1999 World

Health Organization criteria. Using HbA1c criteria there was an increase of 2.2 and 1.4-fold in south Asians and white Europeans detected, respectively. It was concluded that introducing HbA1c ±6.5% as the preferred diagnostic test to diagnose T2DM significantly increased numbers detected with T2DM; however, some people were no longer detected as having T2DM.

STUDY TOPIC

"Comparison of Salivary Glucose and Serum Glucose concentration in Non-Insulin Dependent Diabetes Mellitus patients".

STUDY DESIGN

This is a Case Control type of study.

STUDY DURATION

This study was conducted between March 2010 and April 2011 in the Out Patient Department of Voluntary Helath Services, Adyar, Chennai.

STUDY POPULATION

A total number of 80 patients were involved in the study.

OBTAINING APPROVAL FROM THE AUTHORITIES:

Permission from the ethical committee of **Ragas Dental College and Hospital**, **Chennai** and **Voluntary Health Services**, **Adyar**, **Chennai** was obtained before starting the study.

Due consent to participate in the study was obtained from the Subjects in letter format both in Tamil and English.

MATERIALS

Collection of Saliva Sample:

- A pair of sterile gloves
- Disposable mouth mask.
- Sterile plastic containers for collection of saliva.
- Refrigerator

Collection of Blood Sample:

- Disposable 5ml plastic syringe and 23 gauge needle
- Vacutainer coated with Ethylene diamine tetra acetic acid (EDTA)
- Torniquet
- Sterile Cotton
- 70% alcohol as surface disinfectant
- Sterile vials
- Refrigerator

Equipments:

- Centrifuge for separating plasma from blood
- Semiautoanalyzer (Biosystems BTS-310 Photometer)
- Micro pipette

METHODOLOGY

STUDY GROUP:

The study group comprised of a total number of 80 patients. Out of the 80 patients, 40 were Healthy controls and the other 40 were suffering from Type 2 Diabetes Mellitus.

GROUP I

This study group comprised of 40 Healthy Non Diabetic indiviuals visiting the Out Patient Department at Voluntary Health Services.

Clinical SelectionCriteria:

- Individuals who are apparently healthy with no history of Diabetes, Hypertension and any known systemic diseases.
- Indiviuals with Fasting blood sugar of 125mg/dl or below.
- Indiviuals with Random blood sugar level of 200mg/dl or below.

GROUP II

This study group comprised of 40 patients suffering from Type 2 Diabetes Mellitus visiting the Out Patient Department at Voluntary Health Services.

Clinical Selection Criteria:

- 30 diagnosed Diabetic patients with Type 2 Diabetes Mellitus.
- Patients with fasting blood sugar level of 126mg/dl and above.
- Patients with Random blood sugar level of 200mg/dl and above

INFORMED CONSENT:

Permission from the ethical committee of **Ragas Dental College and Hospital**, **Chennai** and **Voluntary Health Services**, **Adyar**, **Chennai** was obtained before starting the study.

Informed consent was taken from all subjects before including them in the study.

EXCLUSION CRITERIA:

Participants with infectious diseases during one month before saliva sampling, active dental abcesses, and collagen vascular diseases were excluded from the study.

EXAMINATION OF THE SUBJECTS:

The experimental subjects were made to sit comfortably on a chair. Relevant demographic data was collected.An Intra Oral examination is carried out. Whole unstimulated saliva is collected. Saliva samples are collected in sterile test tubes, immediately transferred aseptically to sterile tubes and frozen on dry ice and alcohol. The samples are stored in styroform boxes containing dry ice and carried to a freezer where they are left until time of assessment of the salivary glucose and serum glucose.

SERUM SAMPLE COLLECTION[87]:

Blood samples are taken from the vein in the antecubital fossa. The tourniquet is set around the upper arm of the subject, search for the proper vein by inspecting and palpating and then sterilize the injection site. The vein can be anchored by placing the thumb about two centimeters below the vein and pulling gently to make the skin a little taut. After that, the needle, beveled upward, should be pushed smoothly and quickly into the vein, to minimize the possibility of hemolysis as a result of vascular damage. Immediately after the insertion, the tourniquet should be released to minimize the effect of hemoconcentration. 5 ml of venous blood was drawn and the serum was separated by centrifugation, supernatant was aspirated.

The samples were centrifuged no later than 30 minutes after the sample was drawn.EDTA and Sodium Fluoride were added to prevent the coagulation of blood.All samples were centrifuged at 3000 rpm for 10 min to remove particulate materials and the clean supernatant was proessed immediately for estimation of glucose.

SALIVA SAMPLE COLLECTION[7]:

The subjects were required to abstain from eating for atleast 8 hours. Drinking, smoking or using oral hygiene products was not allowed for at least 1 hour before saliva collection. The patients were asked to rinse their mouth with water and were made to sit comfortably in a chair.Whole unstimulated saliva amounting to 5ml was collected for 5-minutes by spitting method. This was pooled saliva and represented the output from all the salivary glands.

All samples were centrifuged at 3000 rpm for 10 min to remove particulate materials and the clean supernatant was proessed immediately for estimation of glucose.

GLUCOSE ESTIMATION (GOD- POD Method, End Point)[87,88]

INTRODUCTION

Glucose is the reducing monosaccharide that serves as the principal source of cellular energy in the body. It enters into the cell under the influence of insulin and undergoes a series of chemical reactions to produce energy. Lack of insulin or resistance to its action at the cellular level causes Diabetes.Therefore, in Diabetes Mellitus the blood glucose level are very

high. However, high blood glucose level is also observed in the pancreatitis, pituitary or thyroid dysfunction, renal failure and liver disease whereas low glucose level is associated with starvation, hyperinsulinaemia, neopalasms or insulin induced hypoglycemia. Estimation of glucose in serum as well as saliva was done with this method.

PRINCIPLE[89]

Glucose is oxidized by glucose oxidase (GOD) to produce gluconate and hydrogen peroxide. The hydrogen peroxide is then oxidatively coupled with 4 amino- antipyrene (4-AAP) and phenol in the presence of peroxidase (POD) to yield a redquinoeimine dye that is measured at 505nm. The absorbance at 505 nm is proportional to concentration of glucose in the sample.

$$\text{Glucose} + 2H_2O + O_2 \longrightarrow \text{Gluconate} + H_2O_2$$

$$2H_2O + \text{4-AAP} + \text{Phenol} \longrightarrow \text{Quinoeimine Dye}$$

Absorbance of the colored solution is directly proportional tothe glucose concentration, when measured at 505nm.

REAGENT COMPOSITION

Reagent 1:

Glucose Oxidase	20000 u/l
Peroxidase	1200 u/l
4-AAP	0.246 mmol/l

Reagent 2:

Glucose Standard	100 mg/dl

PRECAUTIONS

- Avoid ingestion, do not pipette by mouth.
- Avoid contact with skin and eyes. If spilled, thoroughly wash affected area with water.
- Flush with plenty of water while disposing.

REAGENT

Reagent should be clear. Turbidity and/or precipitation may be because of reagent deterioration.

SAMPLE COLLECTION AND STORAGE

Unhaemolysed serum isto be used for the testing. EDTA and sodium fluoride were the preferred anti-coagulant. Freshly collected samples for assay.

GENERAL ASSAY PARAMETERS

Mode	End Point
Wavelength (nm)	505
Wavelength Range Usable(nm)	500-550
Blank with	Reagent
Sample Volume (µl)	5/10

Reagent R1 (µl)	500/1000
Incubation Time (min)	15 /7
Incubation Temperature (°C)	RT/37
Normal Low (mg/dl)	70
Normal High (mg/dl)	110
Linearity (mg/dl)	Upto 500
Standard Conc. (mg/dl)	100
Units	mg/dl

PROCEDURE

One reagent blank and one standard were used for each assay series.

Pipette into Test Tubes:

Particulars	Blank	Standarad	Sample
Reagent 1	1000µL	1000µL	1000µ
Distilled Water	10µL	-	-
Reagent 2	-	10µL	-
Sample	-	-	10µL

Mix well & incubate for 15 min at room temperature or 7 min at 37^{o}C. Measure the absorbance of standard (A std) and sample (A sample) against reagent blank at 505 nm.

CALCULATION

Glucose concentration is calculated using the following formula:

STATISTICAL ANALYSIS:

All the datas were entered in Microsoft excel sheets. Statistical analysis was done using SPSS software SYSTAT version 7.0.

Mean and standard deviation were estimated in the sample for each study group. Mean values were compared by using one-way ANOVA followed by multiple range tests by Tukey-HSD for multiple group comparison and students 't" test and chi square test for two group comparison.

In the present study $p < 0.05$ was considered as the level of significance.

$$\text{Mean } (X) = \frac{\sum \overline{X}i}{n}$$

$$\text{Standard Deviation} = \sqrt{\frac{\sum(Xi-X)^2}{n-1}}$$

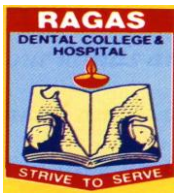
Where Xi is the individual observation and n is the sample size.

ANOVA:

$$\text{F Ratio} = \frac{\text{Variation between observed group averages}}{\text{Variation within each group}}$$

Students "t" test (unpaired)

t=difference in means/standard error of difference



RAGAS DENTAL COLLEGE AND HOSPITAL

2/102 East Coast Road, Uthandi, Chennai – 600019

DEPARTMENT OF ORAL MEDICINE & RADIOLOGY

CASE SHEET PROFORMA

A. GENERAL INFORMATION

S.No: O.P.No: Date:

1. Name:
2. Age:
3. Sex: 1. Male 2. Female:
4. Occupation:
    a. Unemployed
    b. Skilled
    c. Professional
    d. Administration
    e. Trade/Business
    f. Student
5. Address:

6. Income;

    a. <Rs. 1,000/month b. >1,000-5,000/month

    c. >5,000/month

B. History:

1. History relating to Diabetes Mellitus

    a. Age at diagnosis of Diabetes Mellitus

    b. Onset and duration of Diabetes

    c. Family history of Diabetes Mellitus

2. Presence of any other systemic disease

    a. Present b. Absent

    If yes, specify

3. History of medication;

    a. Yes b. No

If yes, specify Duration Name of medicine

INVESTIGATION

1. Random Blood Sugar
2. Salivary Glucose Estimation
3. Fasting Blood Sugar
4. Post Prandial

Figure 1. Armamentarium for Clinical Examination

Figure 2. Autoclave

Figure 3. Centrifuge

Figure 4. Armamentarium for Serum Glucose and Salivary Glucose Estimation

Figure 5. Serum Glucose and Salivary Glucose Estimation kit

Figure 6. Oral Rinse Procedure

The present study is a Case Control study which was conducted in the Out Patient Department of Voluntary Health Services, Adyar, Chennai. It was devised to estimate the Salivary Glucose and Serum Glucose level in Type 2 Diabetes Mellitus patients and Healthy Controls. The study was conducted between March 2010 and April 2011 with 40 Type 2 Diabetes Mellitus patients and 40 Healthy controls.The data obtained from the study was statistically analysed. The results extracted were compared with various variables included in the study and are presented here.

Table 1: Sex wise distribution of subjects in Group I (Healthy Controls)

The study consisted of a total number of 80 subjects. This table denotes the Sex wise distribution of subjects in Group I. Out of the 80 subjects, 40 were included in Healthy Controls (Group I) among whom 16 males were present, accounting for 40% of the group and 24 females accounting for 60% of the group. A significant p value of <.005 was found.

Table 2: Sex wise distribution of subjects in Group II (Type 2 Diabetes Mellitus)

This table denotes the Sex wise distribution of subjects in Group II, 40 subjects were included in Type 2 Diabets Mellitus (Group II) among whom 16 were males accounting for 40% of the population and 24 were

females accounting for 60% of the population. A significant p value of <.005 was found.

Table 3: Age wise distribution of subjects in Group I (Healthy Controls)

This table denotes the Age wise distribution of subjects in Group I. The Age of the subjects included in the study ranged between 28-75 yrs. Among the 40 subjects in Group I, 04 (10%) were below 35yrs, 09 (22.5%) were between 36-45yrs, 13 (32.5%) were between 46-55yrs and finally 14 subjects (35%) were above 55yrs. A significant p value of <.005 was found.

Table 4: Age wise distribution of subjects in Group II (Type 2 Diabetes Mellitus)

This table denotes the Age wise distribution of subjects in Group II. The Age of the subjects included in the study ranged between 28-75 yrs. Among the 40 subjects in Group II, 04 (10%) were below 35yrs, 09 (22.5%) were between 36-45yrs, 13 (32.5%) were between 46-55yrs and 14 (35%) were above 55yrs. A significant p value of <.005 was found.

Table 5: Age and Sex wise distribution of subjects in GroupI (Healthy Controls)

This table shows the distribution of subjects based on Age and Sex in Group I. The Age range was from 28 to 75yrs with the mean age of 50.97 yrs. In the below 35yrs age group there were totally 4 subjects inwhich accounted for 10.0% of the total subjects in the group, with 1 male (6.3%) and 3 females (12.5%) of the total number of males and females

present in the group. Similarly the 36-45yrs age group had 9 subjects in them accounting for 22.5% of the entire group population with 5 males (31.3%) and 4 females (16.7%) of the total males and females present in the group. The 46-55yrs age group comprised of totally 13 patients or 32.5% of the entire group with 5 males (31.3%) and 8 females (33.3%). And finally, the age group above 55yrs comprised of totally 14 patients or 35% of the entire group with 5 males (31.3%) and 9 females (37.5). There is a clear female predilection of 26 (60%) compared to the males who accounted for 14 (40%) of the population in the total sample size of 40 patients. A significant p value of <.005 was found.

Table 6: Age and Sex wise distribution of subjects in GroupII (Type 2 Diabetes Mellitus)

This table shows the distribution of subjects based on Age and Sex in Group II. The Age range was similar to that of Group I with variation between 28 to 75yrs with the Mean Age of 50.97 yrs. The subjects below 35yrs age group totally had 4 subjects in them which accounted for 10.0% of the total subjects in the group, with 1 male (6.3%) and 3 females (12.5%) of the total number of males and females present in this group. Similarly,in the 36-45yrs age group, there were 9 subjects in them accounting for 22.5% of the entire group population with 5 males (31.3%) and 4 females (16.7%) of the total males and females present in the group. The 46-55yrs age group comprised totally of 13 patients or 32.5% of the entire group with 5 males (31.3%) and 8 females (33.3%). And finally the Age group above 55yrs

comprised of totally 14 patients or 35% of the entire group with 5 males (31.3%) and 9 females (37.5). There is a clear female predilection of 26 (60%) compared to the males who accounted for 14 (40%)in the total of 40 patients. A significant p value of <.005 was found.

Table 7: Salivary Glucose level distribution according to Sex in Group I (Healthy Controls)

This table shows the distribution of subjects based on Salivary Glucose levels and Sex in Group I. The Salivary Glucose levels varied from 0–8mg/dl in this group. Totally there were 40 patients among the distribution of 16 males and 24 females. Out of these there were totally 4 subjects ie. 10.0% of the Group I who had a Salivary Glucose level of 0mg/dl out of which 2 were males and 2 were females accounting for 12.5% and 8.3% respectively of the total number of males and females present in this group. Similarly, there were totally 15 subjects ie. 37.5% of the Group I who had a Salivary Glucose level of 1mg/dl out of which 5 were males and 10 were females accounting for 31.3% and 41.7% respectively of the total number of males and females present in the group. There were 11 subjects ie. 27.5% of the Group I who had a Salivary Glucose level of 2mg/dl out of which 4 were males and 7 were females accounting for 25.0% and 29.2% respectively of the total number of males and females present in the group. There were 5 subjects ie. 12.5% of the Group I who had a Salivary Glucose level of 3mg/dl out of which 2 were males and 3 were females accounting for 12.5% and 12.5% respectively of the total number of males and females

present in this group. There were 2 subjects ie. 12.5% of the Group I who had a Salivary Glucose level of 4mg/dl out of which 2 were males and 0 were females accounting for 12.5% and 0% respectively of the total number of males and females present in the group. There was 1 subject ie. 2.5% of the Group I who had a Salivary Glucose level of 5mg/dl out of which 1 was male and there were 0 females accounting for 6.3% and 0% respectively of the total number of males and females present in this group. There was 1 subject ie. 2.5% of the Group I who had a Salivary Glucose level of 7mg/dl out of which there were 0 males and 1 female accounting for 0% and 4.2% respectively of the total number of males and females present in this group. Lastly, there was 1 subject ie. 2.5% of the Group I who had a Salivary Glucose level of 8mg/dl out of whom there were 0 males and 1 female accounting for 0% and 4.2% respectively of the total number of males and females present in this group. An insignificnat p value of >.005 was found.

Table 8: Salivary Glucose level distribution according to Sex in Group II (Type 2 Diabetes Mellitus)

This table shows the distribution of subjects based on Salivary Glucose levels and Sex in Group II. The Salivary Glucose levels varied from 1–5mg/dl in this group. Totally, there were 40 patients among the distribution of 16 males and 24 females. Out of which there were totally 9 subjects ie. 22.5% of the Group II who had a Salivary Glucose level of 1mg/dl out of which 3 were males and 6 were females accounting for 18.8% and 25.0% of the total number of males and females present in this group.

Similarly, there were totally 19 subjects ie. 47.5% of the Group II who had a Salivary Glucose level of 2mg/dl out of which 8 were males and 11 were females accounting for 50.0% and 45.8% respectively of the total number of males and females present in this group. There were 5 subjects ie. 12.5% of the Group II who had a Salivary Glucose level of 3mg/dl out of which there was 1 male and 4 females accounting for 6.3% and 16.7% respectively of the total number of males and females present in this group. There were 4 subjects ie. 10.0% of the Group I who had a Salivary Glucose level of 4mg/dl out of which 2 were males and 2 were females accounting for 12.5% and 8.3% respectively of the total number of males and females present in this group. Finally, there were 3 subjects ie. 7.5% of the Group II who had a Salivary Glucose level of 5mg/dl out of which 2 were males and 1 was a female accounting for 12.5% and 4.2% respectively of the total number of males and females present in this group. An insignificant p value of >.005 was found.

Table 9: Salivary Glucose distribution according to Age in Group I (Healthy Controls)

This table shows the distribution of subjects based on Salivary Glucose levels and their Age in Group I. The Salivary Glucose levels varied from 0 – 8mg/dl in this group. Salivary Glucose level of 0mg/dl was found in totally 4 subjects comprising 10% of the total subjects out of whom there was 1 subject (25.0%) totally out of the Below 35yrs age group, 0 (0%) in the 36-45yrs age group, 2 (15.4%) in age group 46-55yrs and 1 (7.1%) age

group above 55yrs. Similarly, the Salivary Glucose level of 1mg/dl was found totally in 15 subjects comprising of 37.5% out of whom there were 3 subjects (75.0%)totally who belonged to the below 35yrs age group, 4 (44.4%) in 36-45yrs age group, 2 (15.4%) in 46 – 55yrs age group and 6 (42.9%) in above 55yrs age group. Salivary Glucose level of 2mg/dl was found in 11 patients accounting for 27.5% of the entire group totally out of which 0 (0%) subjects below 35yrs age group, 3 (33.3%) in 36-45yrs age group, 6 (46.2%) in 46 – 55yrs age group and 2 (14.3%) in above 55yrs age group.Salivary Glucose level of 3mg/dl was found totally in 5 subjects accounting for 12.5% of the entire group, out of which 0 (0%) subjects were below 35yrs age group, 1 (11.1%) in 36-45yrs age group, 0 (0%) in 46–55yrs age group and 4 (28.6%) in above 55yrs age group.Salivary Glucose level of 4mg/dl was found to be present in totally 2 subjects accounting for 5.0% of the groups with 0 subjects below 35yrs age group, 0 in 36-45yrs age group, 2 (15.4%) in 46–55yrs age group and 0 (0%) in above 55yrs age group. Salivary glucose level of 5mg/dl was found in only 1 subject accounting for 2.5% of the population with 0 (0%) subject below 35yrs age group, only 1 (11.1%) in 36-45yrs age group, 0 (0%) in 46–55yrs age group and 0 (0%) in above 55yrs age group.Salivary Glucose level of 7mg/dl was found in only 1 subject accounting for 2.5% of the entire group with 0 (0%) subjects present in the below 35yrs age group, 0 (0%) in 36-45yrs age group, 1 (7.7%) in 46 – 55yrs age group and 0 (0%) in the above 55yrs age group. Finally, Salivary Glucose level of 8mg/dl was

found in only 1 patient accounting for 2.5% of the entire group with 0 (0%) subjects below 35yrs age group, 0 (0%) in 36-45yrs age group, 0 (0%) in 46–55yrs age group and 1 (7.1%) in above 55yrs age group. Hence, of the 40 patients in this group, 4 (10.0%) were among the <35yrs age group, 9 (22.5%) in the 36-45yrs age group, 13 (32.5%) in the 46-55yrs age group and finally 14 (35.0%) in the >55yrs age group. Insignificant p value of >.005 was found.

Table 10:Salivary Glucose level distribution according to Age in Group II (Type II Diabetes Mellitus)

This table shows the distribution of subjects based on Salivary Glucose levels and Age in Group II. The Salivary Glucose levels varied from 1 – 5mg/dl in this group. Salivary Glucose level of 1mg/dl was found in totally 9 (22.5%) subjects of the entire group among whom 0 (0%) subjects were there in the below 35yrs age group, 3 (33.3%) in the 36-45yrs age group, 3 (23.1%) in 46-55yrs age group and 3 (21.4%) in the age group above 55yrs. Similarly, the Salivary Glucose level of 2mg/dl was found in totally 19 (47.5%) patients in the entire group among whom 3 (75%)subjects were in the below 35yrs age group, 3 (33.3%) in 36-45yrs age group, 6 (46.2%) in 46 – 55yrs age group and 7 (50%) in above 55yrs age group. Salivary Glucose level of 3mg/dl was found totally in 5 subjects accounting for 12.5% of the entire group out of which 0 (0%) subjects were present in the below 35yrs age group, 2 (22.2%) in 36-45yrs age group, 2 (15.4%) in 46 – 55yrs age group and 1 (7.1%) in above 55yrs age group. Salivary

Glucose level of 4mg/dl was found in totally 4 subjects among accounting for 10.0% of the population among whom1 (25.0%) subject was in the below 35yrs age group, 0 (0%) in 36-45yrs age group, 1 (7.7%) in 46 – 55yrs age group and 2 (14.3%) in above 55yrs age group. Salivary glucose level of 5mg/dl was found totally in 3 subjects accounting for 7.5% of the group with 0 (0%) subjects below 35yrs age group, 1 (11.1%) in 36-45yrs age group, 1 (7.7%) in 46 – 55yrs age group and lastly 1 (7.1%) in above 55yrs age group.Hence, of the 40 patients in this group, 4 (10.0%) were among the <35yrs age group, 9 (22.5%) in the 36-45yrs age group, 13 (32.5%) in the 46-55yrs age group and finally 14 (35.0%) in the >55yrs age group. The p value was found to be insignificant at >.005.

Table 11: Fasting Blood Glucose distribution according to Sex in Group I (Healthy Controls)

This table shows the distribution of Fasting blood glucose in males and females in Group I.The subjects among this group were Healthy Non Diabetic controls who had a Fasting Blood Glucose of <125mg/dl with a distribution of 16 males and 24 females. The Mean Fasting Blood Glucose was found to be 101.63mg/dl in males and 93.54mg/dl in females in this group.A significant p value < .005 was found.

Table 12: Fasting Blood Glucose level distribution according to sex in Group II (Type 2 Diabetes Mellitus)

This table shows the distribution of Fasting Blood Glucose in males and females in Group II.The subjects among this group were known cases of

Type II Diabetes with a Fasting Blood Glucose of >125 mg/dl and comprised of 16 males and 24 females. The Mean Fasting Blood Glucose was found to be 208.13mg/dl in males and 197.67mg/dl in females in this group. A significant p value of <.005 was found.

Table 13: Fasting Blood Glucose level distribution according to Age in Group I (Healthy Controls)

This table shows the distribution of Fasting Blood Glucose <125 mg/dl according to Age in Group I. There were 4 subjects present in the <35yrs age group, with a mean Fasting Blood glucose of 79mg/dl, 9 subjects in the 36-45yrs age group with a mean Fasting Blood Glucose of 100.22mg/dl, with another 13 patients in the age group between 46-55yrs with a mean of 94.08mg/dl and finally 14 patients in the age group above 55yrs with a mean Fasting Blood Glucose of 102.00mg/dl. The mean Fasting Blood Glucose among the entire group was 96.78mg/dl. A significant p value of <.005 was found.

Table 14: Fasting Blood Glucose level distribution according to Age in Group II (Type 2 Diabetes Mellitus)

This table shows the distribution of Fasting Blood Glucose of >125mg/dl according to age in Group II.There were 4 subjects present in the <35yrs age group, with a Mean Fasting Blood Glucose of 179 mg/dl, 9 subjects in the 36-45yrs age group with a mean of 236.67 mg/dl, with another 13 patients in the age group between 46-55yrs with a mean of 208.92 mg/dl and finally 14 patients in the age group above 55yrs with a

Mean Fasting Blood Glucose of 179.43 mg/dl. The mean Fasting Glucose among this group was 201.85 mg/dl. A significant p value of <.005 was found.

Table 15: Multiple comparisons between Age and Fasting Blood Glucose in Group I (Healthy Controls)

This table shows the multiple significant comparisons that were found between Age and Fasting Blood Glucose values in Group I (Healthy Controls). On comparison of the subjects in the below 35yrs age group with the subjects in the 36-45yrs age group a mean difference of (-)20.72 was found and an insignificant p value of >.005. On comparison of the subjects in the below 35yrs age group with those in the 46-55yrs a mean difference of (-) 14.58 was found with an insignificant p value of >.005. The comparison of the subjects in the below 35yrs age group with the subjects in the above 55yrs age group a mean difference of (-)22.50 and a significant p value of < .005 was found. On comparing the subjects in the 36-45yrs age group with the subjects in the below 35yrs age group a mean difference of 20.72 and an insignificant p value of >.005 was found. On comparing the 36-45yrs age group with the subjects in the 46-55yrs a mean difference of 6.15 and an insignificant p value of >.005. On comparing the subjects in the 36-45yrs age group with those in the above 55yrs age group a mean difference of (-) 1.78 and an insignificant p value of >.005 was found. On comparing the subjects in the 46-55yrs age group with the subjects in the below 35yrs age group a mean difference of 14.58 and an insignificant p

value of >.005 was found. On comparing the subjects in the 46-55yrs age group with the subjects in the 36-45yrs age group a mean difference of (-) 6.15 and an insignificant p value>.005 was found. On comparing the subjects in the 46-55yrs age group with the subjects in the above 55yrs a mean difference of (-)7.92 and an insignificant p value of >.005 was found. On comparing the subjects in the above 55yrs age group with the subjects in the below 35yrs a mean difference of 22.50 and a significant p value of < .005 was found. On comparing the subjects in the above 55yrs age group with the subjects in the 36-45yrs age group a mean difference of 1.78 and an insignificant p value of >.005 was found. And finally, on comparing the subjects in the above 55yrs age group with the subjects in the 46-55yrs a mean difference of 7.92 and an insignificant p value of >.005 was found.

Table 16: Correlation between Fasting Blood Gluose and Salivary Glucose in Group I (Healthy Controls)

This table shows the corelation between the Fasting Blood Glucose (<125 mg/dl) and the Salivary Glucose values in Group I (Healthy control). The Pearson Correlation was utilized. A p value in this group of 40 subjects was found to be >.005 which is insignificant.

Table 17: Corelation between Fasting blood glucose and Salivary Glucose in Group II (Type 2 Diabetes Mellitus)

This table shows the corelation between the Fasting Blood Glucose (>125 mg/dl) and the Salivary Glucose levels in Group II (Type 2

Diabetics). The Pearson Correlation was utilized. An insignificant p value of >.005 was found in this group of 40 subjects.

Table 18: Correlation between Salivary and Fasting Blood Glucose levels in Group I and II.

This table shows the correlation between the Salivary and Fasting Blood Glucose levels among the Healthy controls and the Type 2 Diabetes Mellitus subjects. The Pearson Correlation was utilized. An insignifiant p value of >.005 was found in the total sample of 80 subjects.

Table 1: Sex Wise Distribution of Subjects in Group I
(Healthy Controls)

VALID	FREQUENCY	PERCENT (%)
MALE	16	40.0%
FEMALE	24	60.0%
TOTAL	40	100.0%

p value < .005 (Significant)

Table 2: Sex Wise Distribution of Subjects in Group II
(Type 2 Diabetes Mellitus)

VALID	FREQUENCY	PERCENT (%)
MALE	16	40.0%
FEMALE	24	60.0%
TOTAL	40	100.0%

p value < .005 (Significant)

Table 3: Age Wise Distribution of Subjects in Group I

(Healthy Controls)

VALID	BELOW 35yrs	36-45 yrs	46-55 yrs	ABOVE 55 yrs	TOTAL
FREQUENCY	4	9	13	14	40
PERCENT (%)	10.0%	22.5%	32.5%	35.0%	100.0%

p value < .005 (Significant)

Table 4: Age Wise Distribution of Subjects in Group II

(Type 2 Diabetes Mellitus)

VALID	BELOW 35 yrs	36-45 yrs	46-55 yrs	ABOVE 55 yrs	TOTAL
FREQUENCY	4	9	13	14	40
PERCENT (%)	10.0%	22.5%	32.5%	35.0%	100.0%

p value < .005 (Significant)

Table 5: Age and Sex Wise Distribution of Subjects in Group I

(Healthy Controls)

AGE IN YEARS	SEX		TOTAL
	MALE	FEMALE	
BELOW 35yrs	1	3	4
	6.3%	12.5%	10.0%
36-45yrs	5	4	9
	31.3%	16.7%	22.5%
46-55yrs	5	8	13
	31.3%	33.3%	32.5%
ABOVE 55yrs	5	9	14
	31.3%	37.5%	35.0%
TOTAL	16	24	40
	40.0%	60.0%	100.0%

Age range – 28 -75 yrs Mean age – 50.97yrs

p value <.005 (Significant)

Table 6: Age and Sex Wise Distribution of Subjects in Group II (Type 2 Diabetes Mellitus)

AGE IN YEARS	SEX		TOTAL
	MALE	FEMALE	
BELOW 35yrs	1	3	4
	6.3%	12.5%	10.0%
36-45yrs	5	4	9
	31.3%	16.7%	22.5%
46-55yrs	5	8	13
	31.3%	33.3%	32.5%
ABOVE 55yrs	5	9	14
	31.3%	37.5%	35.0%
TOTAL	16	24	40
	40.0%	60.0%	100.0%

Age range – 28 – 75 yrs Mean age – 50.97 yrs

p value <.005 (Significant)

Table 7: Salivary Glucose Distribution according to Sex in Group I (Healthy Controls)

Salivary Glucose mg/dl	Sex		Total
	Male	Female	
0 mg/dl	2	2	4
	12.5%	8.3%	10.0%
1 mg/dl	5	10	15
	31.3%	41.7%	37.5%
2 mg/dl	4	7	11
	25.0%	29.2%	27.5%
3 mg/dl	2	3	5
	12.5%	12.5%	12.5%
4 mg/dl	2	0	2
	12.5%	.0%	5.0%
5 mg/dl	1	0	1
	6.3%	.0%	2.5%
7 mg/dl	0	1	1
	.0%	4.2%	2.5%
8 mg/dl	0	1	1
	.0%	4.2%	2.5%
Total	16	24	40
	40.0%	60.0%	100.0%

p value > .005 (Insignificant)

Table 8: Salivary Glucose Distribution according to Sex in Group II

(Type 2 Diabetes Mellitus)

Salivary Glucose mg/dl	Sex		Total
	Male	Female	
1 mg/dl	3	6	9
	18.8%	25.0%	22.5%
2 mg/dl	8	11	19
	50.0%	45.8%	47.5%
3 mg/dl	1	4	5
	6.3%	16.7%	12.5%
4 mg/dl	2	2	4
	12.5%	8.3%	10.0%
5 mg/dl	2	1	3
	12.5%	4.2%	7.5%
Total	16	24	40
	40.0%	60.0%	100.0%

p value >.005 (Insignificant)

Table 9: Salivary Glucose Distribution according to Age in Group I (Healthy Controls)

Salivary Glucose mg/dl	Age in years				Total
	Below 35yrs	36-45 yrs	46-55 yrs	Above 55yrs	
0 mg/dl	1	0	2	1	4
	25.0%	.0%	15.4%	7.1%	10.0%
1 mg/dl	3	4	2	6	15
	75.0%	44.4%	15.4%	42.9%	37.5%
2 mg/dl	0	3	6	2	11
	.0%	33.3%	46.2%	14.3%	27.5%
3 mg/dl	0	1	0	4	5
	.0%	11.1%	.0%	28.6%	12.5%
4 mg/dl	0	0	2	0	2
	.0%	.0%	15.4%	.0%	5.0%
5 mg/dl	0	1	0	0	1
	.0%	11.1%	.0%	.0%	2.5%
7 mg/dl	0	0	1	0	1
	.0%	.0%	7.7%	.0%	2.5%
8 mg/dl	0	0	0	1	1
	.0%	.0%	.0%	7.1%	2.5%
Total	4	9	13	14	40
	10.0%	22.5%	32.5%	35.0%	100.0%

p value > .005 (Insignificant)

Table 10: Salivary Glucose distribution according to Age in Group II (Type 2 Diabetes Mellitus)

Salivary Glucose mg/dl	AGE IN YEARS				TOTAL
	Below 35yrs	36-45 yrs	46-55 yrs	Above 55yrs	
1 mg/dl	0	3	3	3	9
	.0%	33.3%	23.1%	21.4%	22.5%
2 mg/dl	3	3	6	7	19
	75.0%	33.3%	46.2%	50.0%	47.5%
3 mg/dl	0	2	2	1	5
	.0%	22.2%	15.4%	7.1%	12.5%
4 mg/dl	1	0	1	2	4
	25.0%	.0%	7.7%	14.3%	10.0%
5 mg/dl	0	1	1	1	3
	.0%	11.1%	7.7%	7.1%	7.5%
Total	4	9	13	14	40
	10.0%	22.5%	32.5%	35.0%	100.0%

p value > .005 (Insignificant)

Table 11: Fasting Serum Glucose distribution according to Sex in Group I (Healthy Controls)

Fasting Blood Sugar mg/dl	Sex	N	Mean
<125 mg/dl	Male	16	101.63
<125 mg/dl	Female	24	93.54

p value <.005 (Significant)

Table 12: Fasting Serum Glucose distribution according to Sex in Group II (Type 2 Diabetes Mellitus)

Fasting Blood Sugar mg/dl	Sex	N	Mean
>125 mg/dl	Male	16	208.13
>125 mg/dl	Female	24	197.67

p value <.005 (Significant)

Table 13: Fasting Blood Glucose (<125 Mg/dl) distribution according to Age in Group I (Healthy Controls)

AGE	BELOW 35yrs	36-45 yrs	46-55 yrs	ABOVE 55yrs	TOTAL
N	4	9	13	14	40
MEAN (mg/dl)	79.00	100.22	94.08	102.00	96.78

p value <.005 (Significant)

Table 14: Fasting Serum Glucose distribution according to Age in Group II (Type 2 Diabetes Mellitus)

AGE	BELOW 35yrs	36-45 yrs	46-55 yrs	ABOVE 55yrs	TOTAL
N	4	9	13	14	40
MEAN (mg/dl)	179.00	236.67	208.92	179.43	201.85

p value<.005 (Significant)

Table 15: Multiple Comparisons between Age and Fasting Blood Glucose in Group I (Healthy Controls)

(I) Age in years	(J) Age in years	Mean Difference (I-J)	Sig.
Below 35yrs	36-45 yrs	-20.72(*)	.018
	46-55 yrs	-14.58	.117
	Above 55yrs	-22.50(*)	.005
36-45 yrs	Below 35yrs	20.72(*)	.018
	46-55 yrs	6.15	.582
	Above 55 yrs	-1.78	.982
46-55 yrs	Below 35yrs	14.58	.117
	36-45 yrs	-6.15	.582
	Above 55yrs	-7.92	.265
Above 55 yrs	Below 35yrs	22.50(*)	.005
	36-45 yrs	1.78	.982
	46-55 yrs	7.92	.265

Table 16: Correlation between Fasting Blood Glucose and Salivary Glucose in Group I (Healthy Controls)

	Fasting Blood Sugar mg/dl	Salivary Glucose mg/dl
Pearson Correlation	1	0.083
Sig. (2-tailed)	-	0.610
N	40	40
Pearson Correlation	0.083	1
Sig. (2-tailed)	0.610	-
N	40	40

p value >.005 (Insignificant)

Table 17: Correlation between Fasting Blood Glucose and Salivary Glucose in Group II (Type 2 Diabetes Mellitus)

	Fasting Blood Sugar mg/dl	Salivary Glucose mg/dl
Pearson Correlation	1	0.118
Sig. (2-tailed)	-	0.468
N	40	40
Pearson Correlation	0.118	1
Sig. (2-tailed)	0.468	-
N	40	40

p value >.005 (Insignificant)

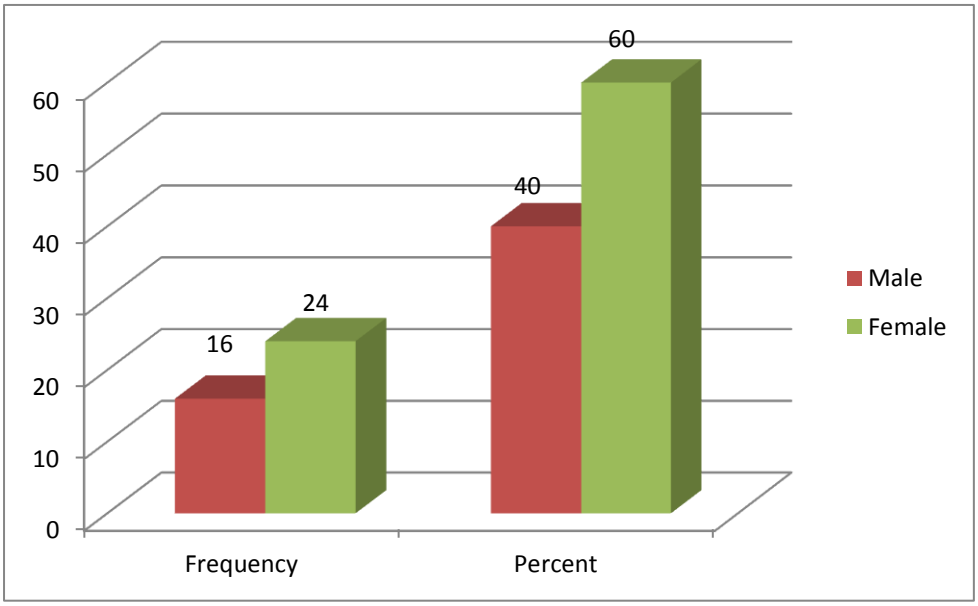
Table 18: Correlation between Salivary and Serum Glucose levels

	Fasting Blood Sugar mg/dl	Salivary Glucose mg/dl
Pearson Correlation	1	0.136
Sig. (2-tailed)	-	0.228
N	80	80
Pearson Correlation	0.136	1
Sig. (2-tailed)	0.228	-
N	80	80

p value > .005 (Insignificant)

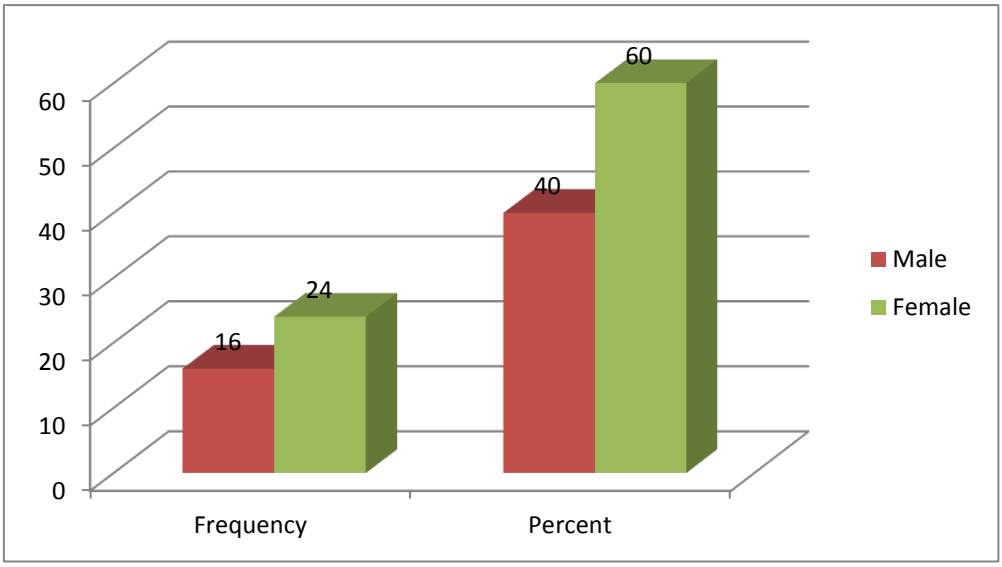
Graph 1: Sex Wise Distribution of Subjects in Group I

(Healthy Controls)



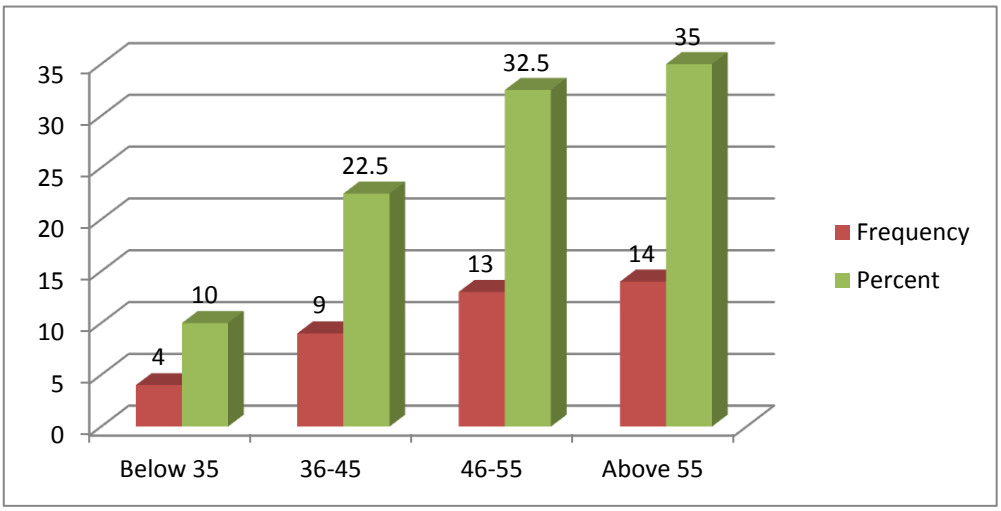
Graph 2: Sex Wise Distribution of Subjects in Group II

(Type 2 Diabetes Mellitus)



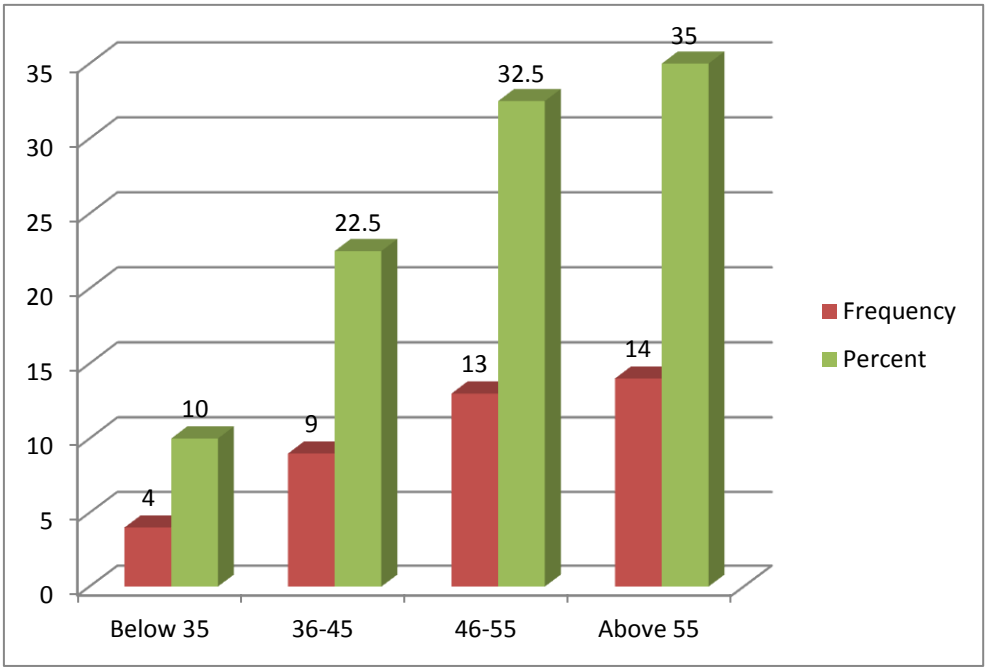
Graph 3: Age Wise Distribution of Subjects in Group I

(Healthy Controls)



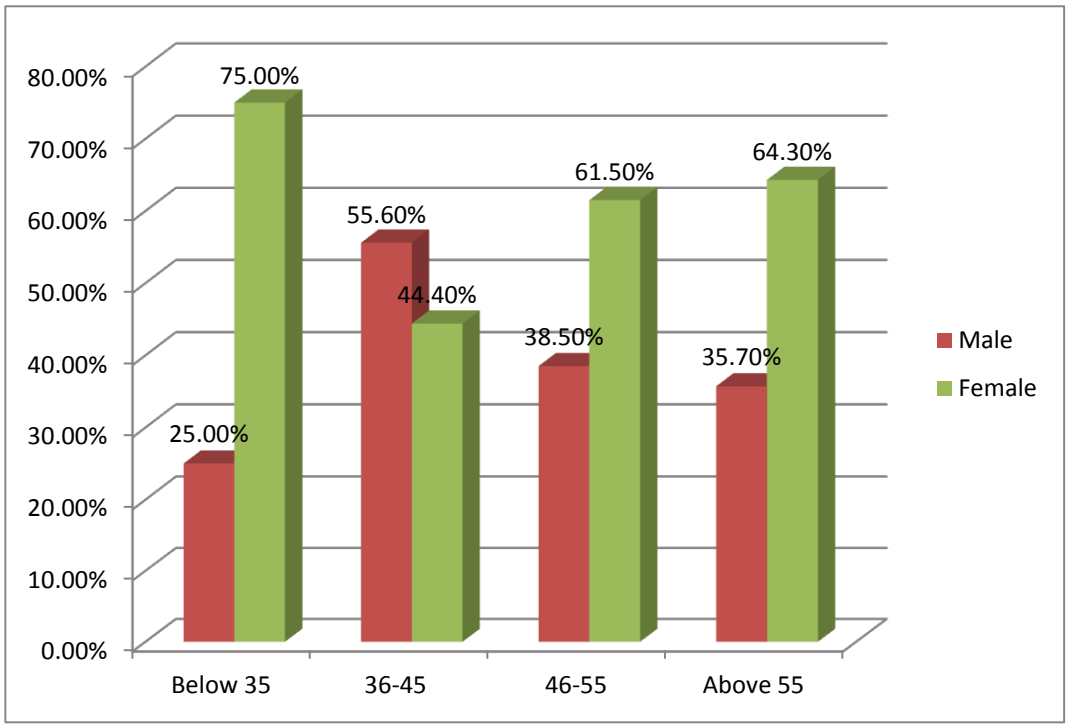
Graph 4: Age Wise Distribution of Subjects in Group II

(Type 2 Diabetes Mellitu)

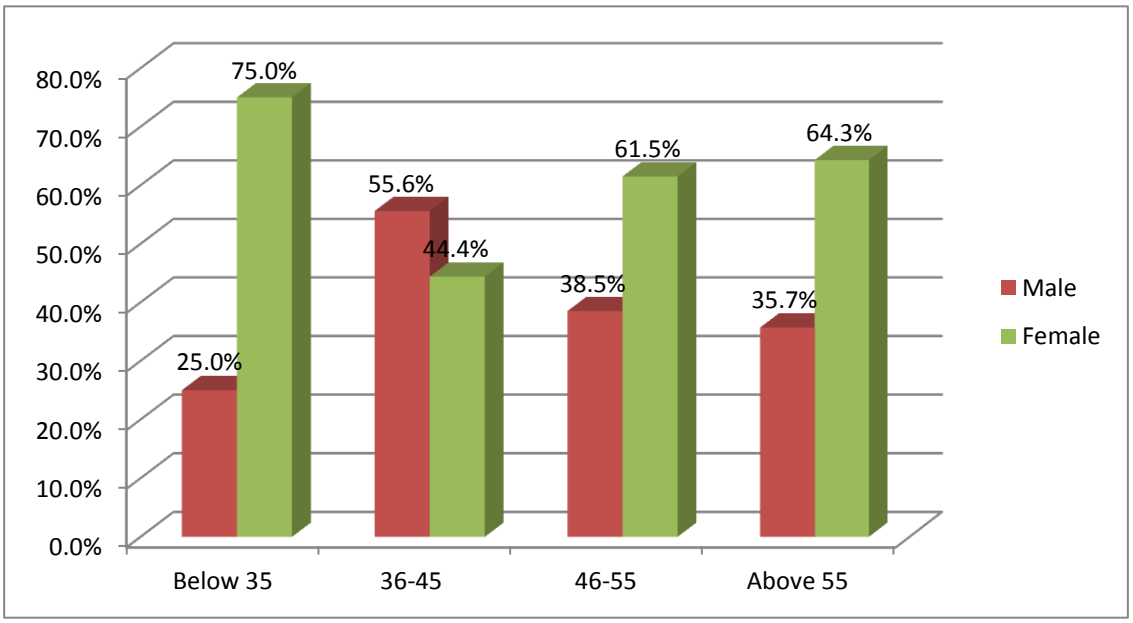


Graph 5: Age and Sex Wise Distribution of Subjects in Group I

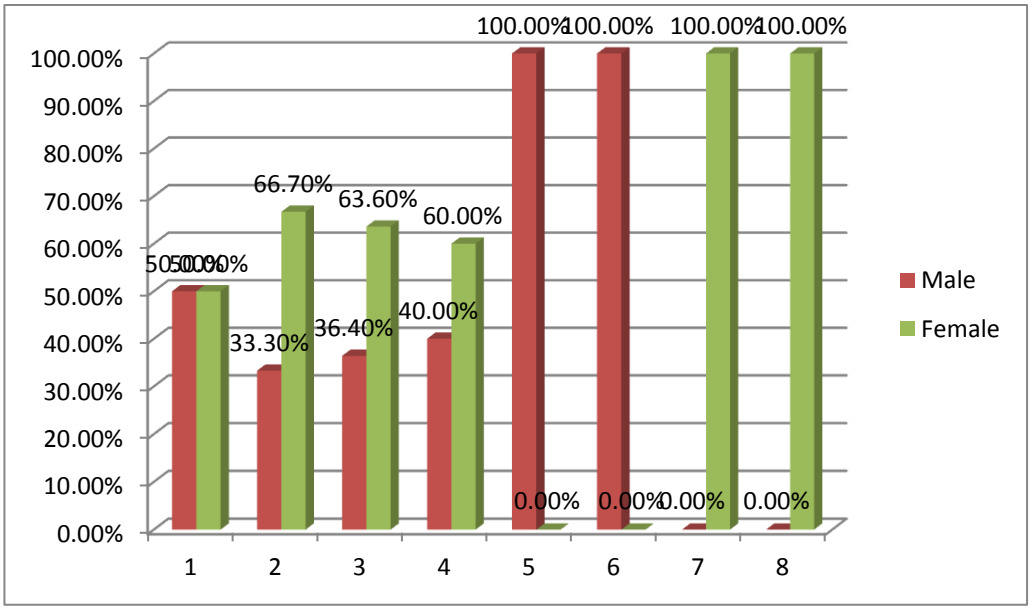
(Healthy Controls)



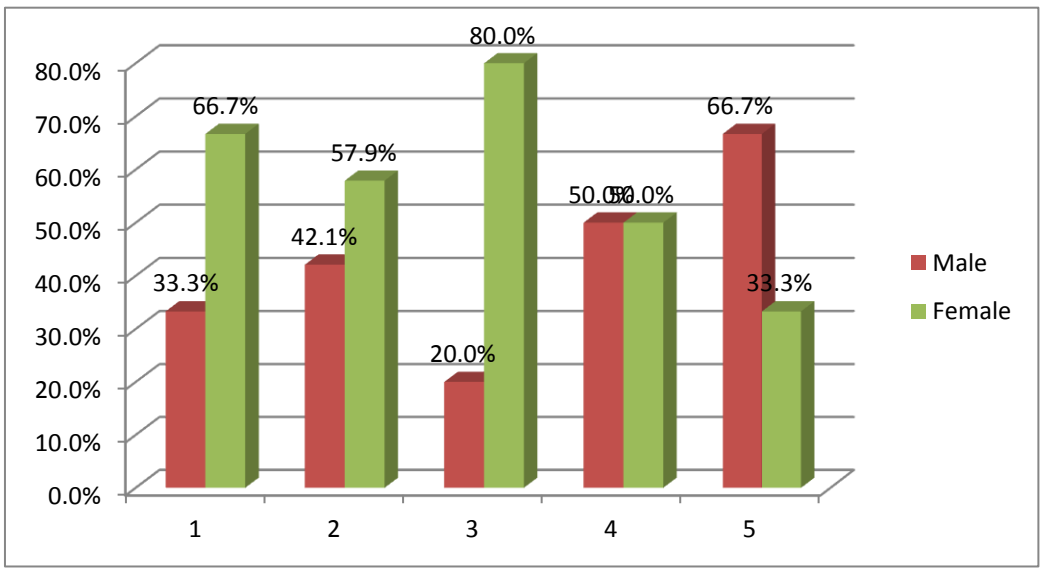
Graph 6: Age and Sex Wise Distribution of Subjects in Group II
(Type 2 Diabetes Mellitus)



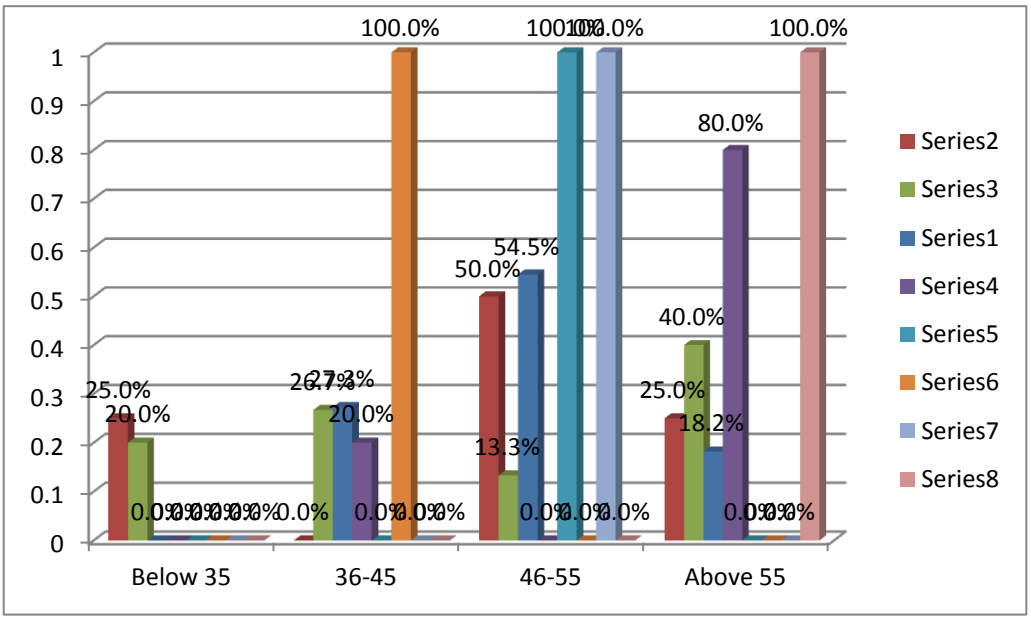
Graph 7: Salivary Glucose Distribution According to Sex in Group I (Healthy Controls)



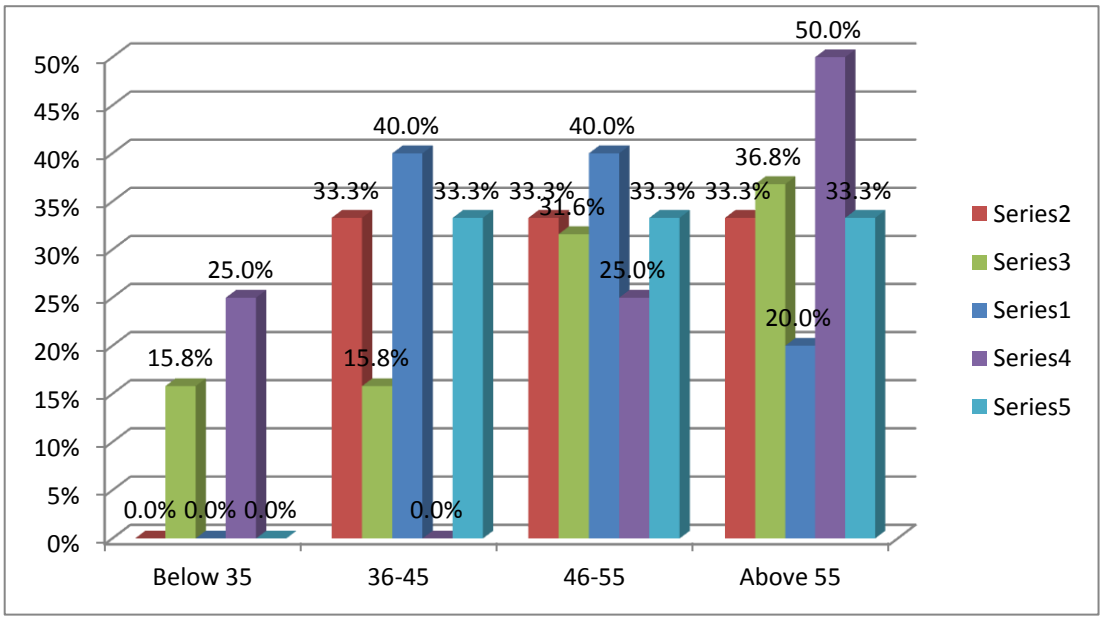
Graph 8: Salivary Glucose Distribution According to Sex in Group II (Type 2 Diabetes Mellitus)



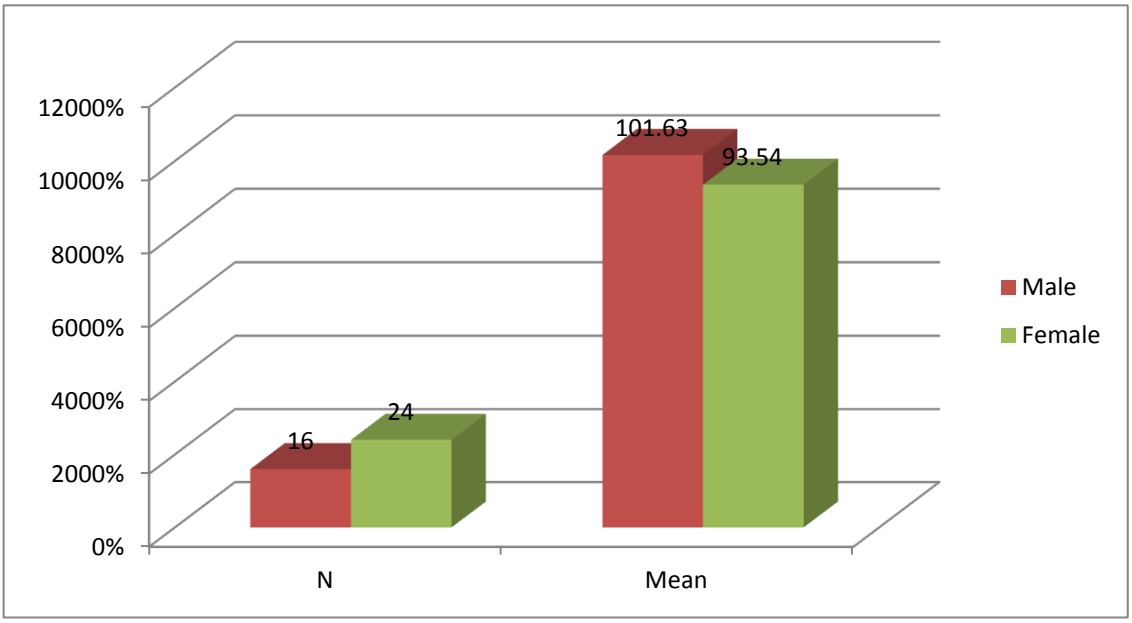
Graph 9: Salivary Glucose Distribution According to Age in Group I (Healthy Controls)



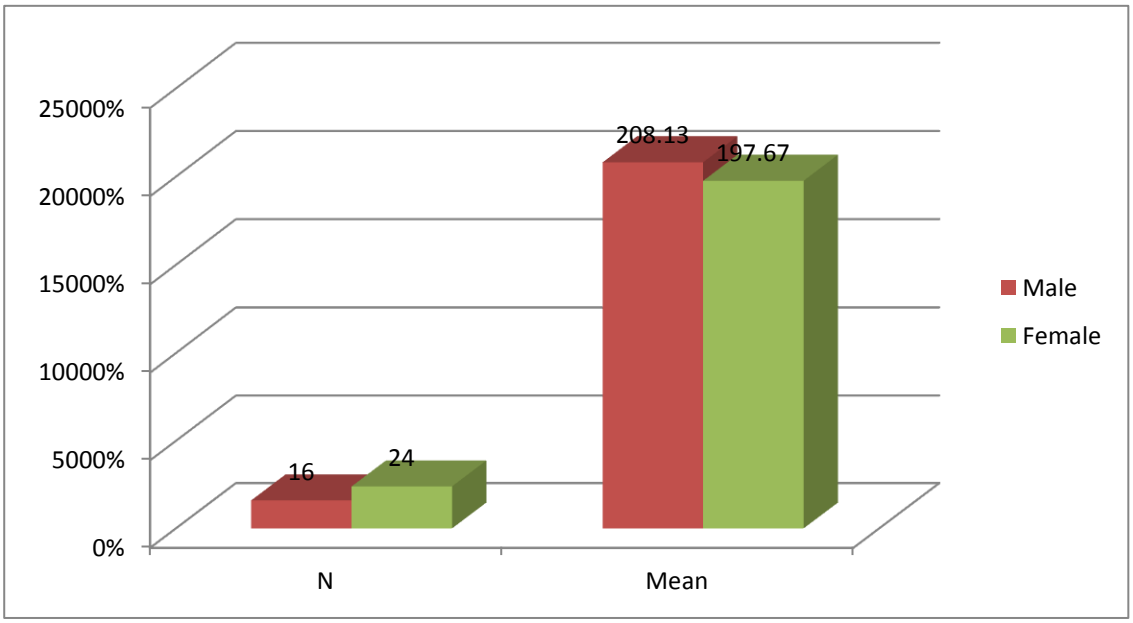
Graph 10: Salivary Glucose Distribution According to Age in Group II (Type 2 Diabetes Mellitus)



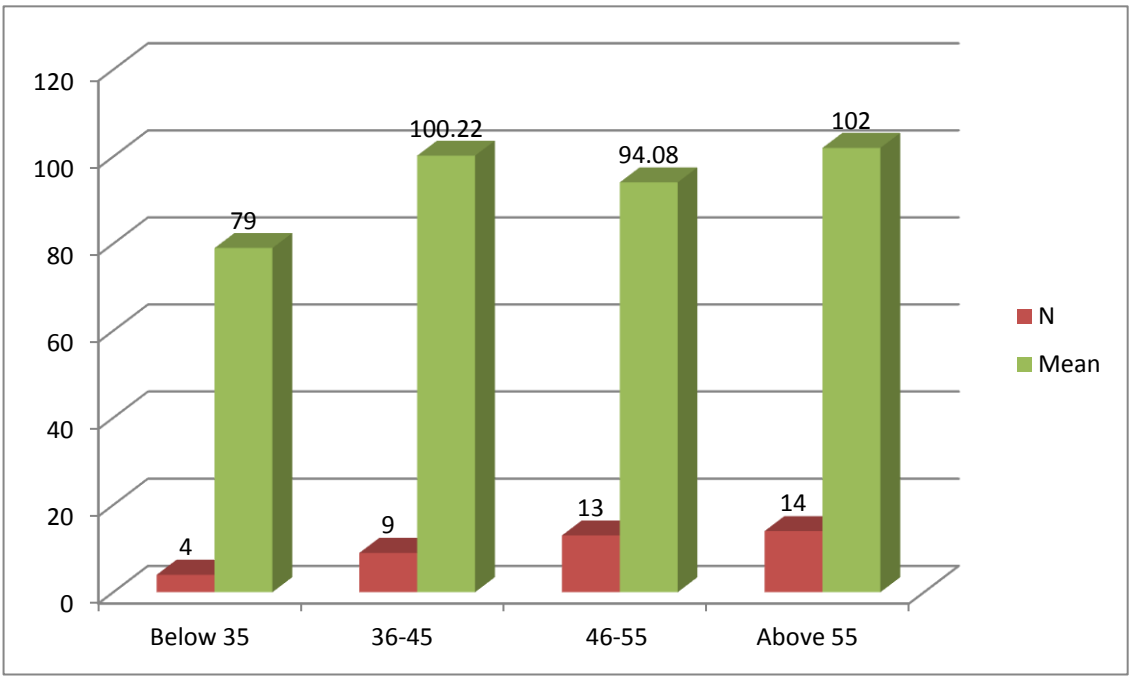
Graph 11: Fasting Serum Glucose Distribution According to Sex in Group I (Healthy Controls)



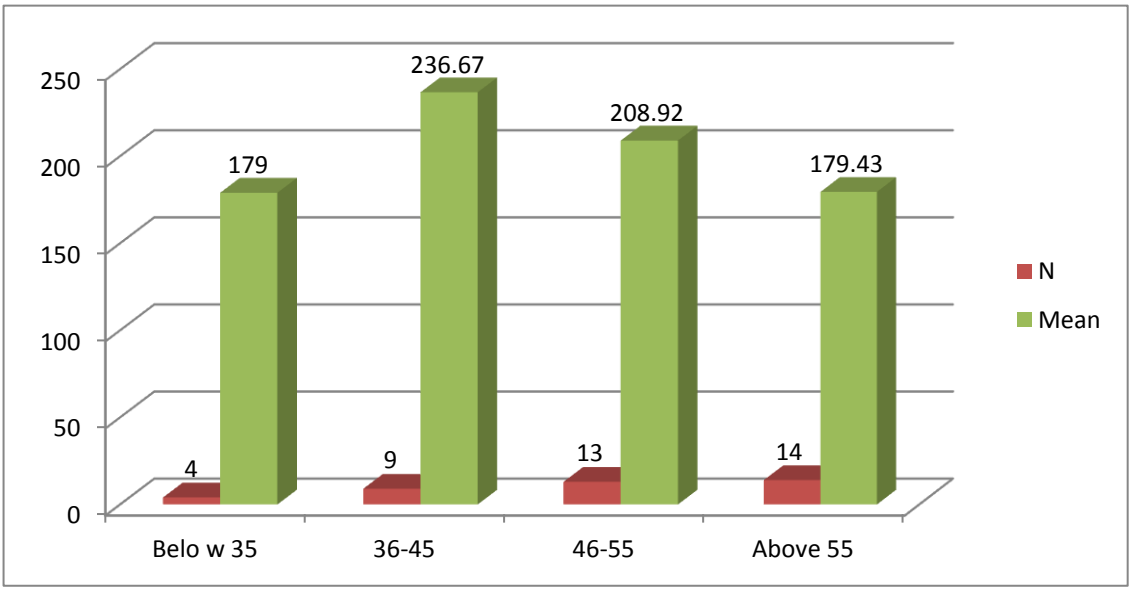
Graph 12: Fasting Serum Glucose Distribution According to Sex in Group II (Type 2 Diabetes Mellitus)



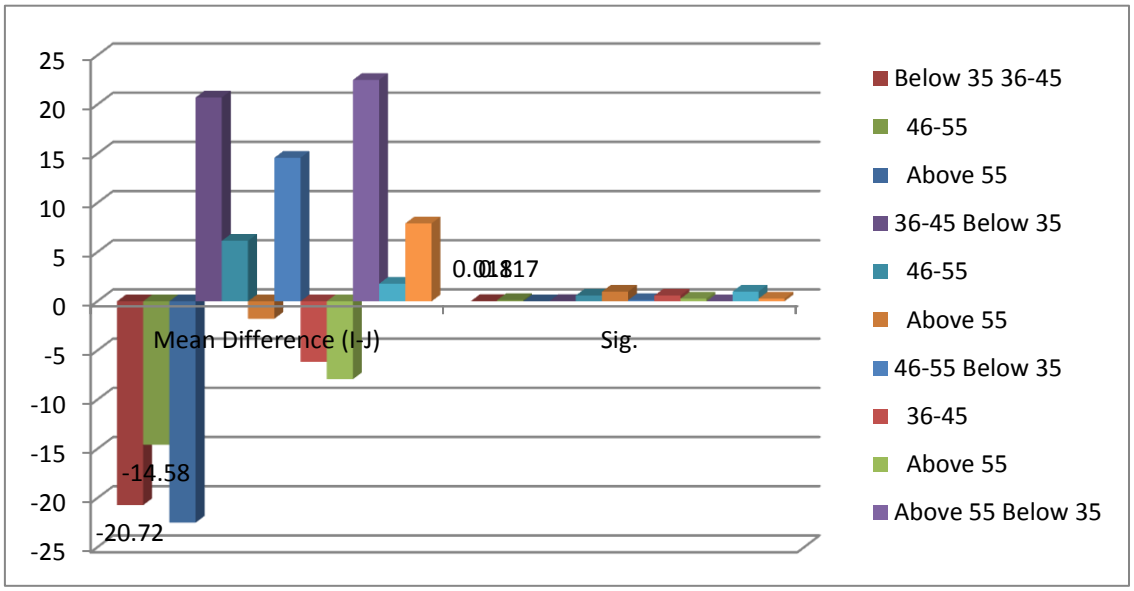
Graph 13: Fasting Blood Glucose Distribution According to Age in Group I (Healthy Controls)



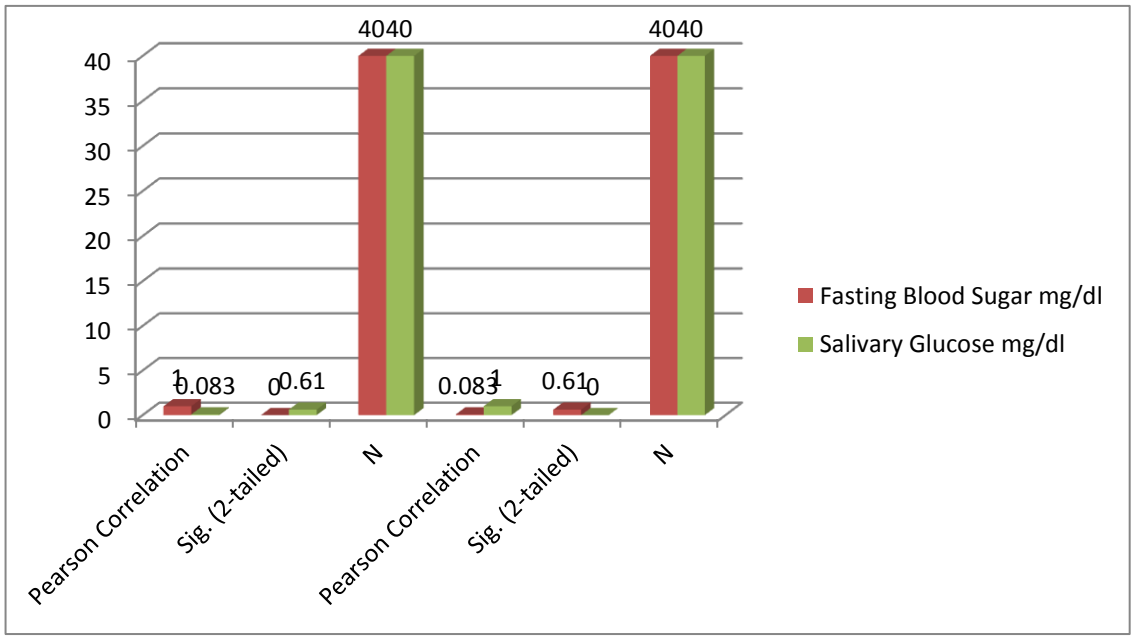
Graph 14: Fasting Serum Glucose Distribution According to Age in Group II (Type 2 Diabetes Mellitus)



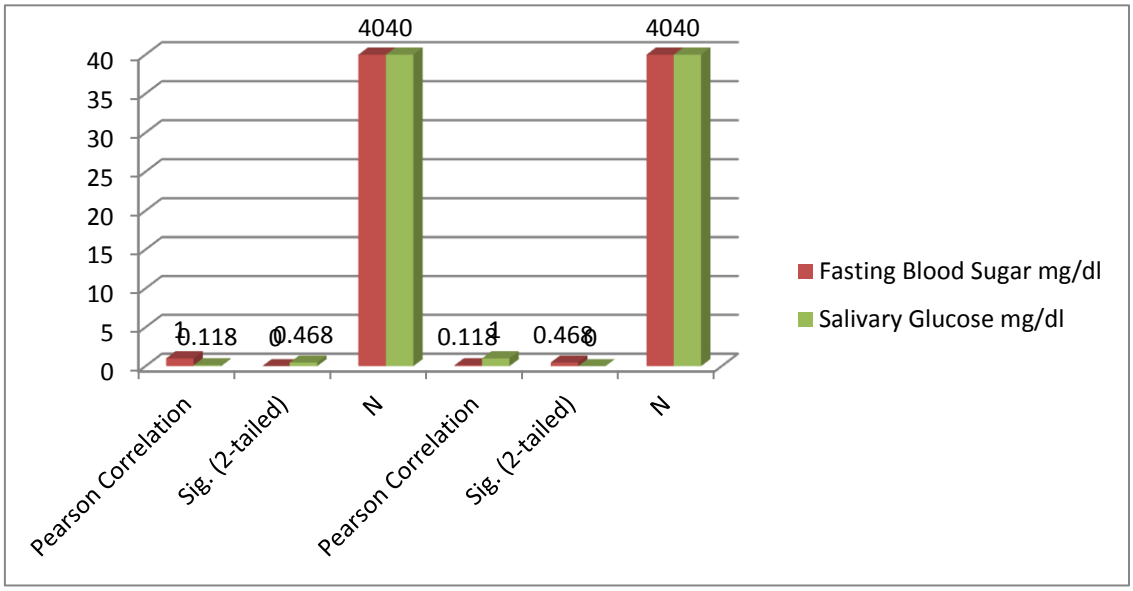
Graph 15: Multiple Comparison Between Age and Fasting Blood Glucose in Group I (Healthy Controls)



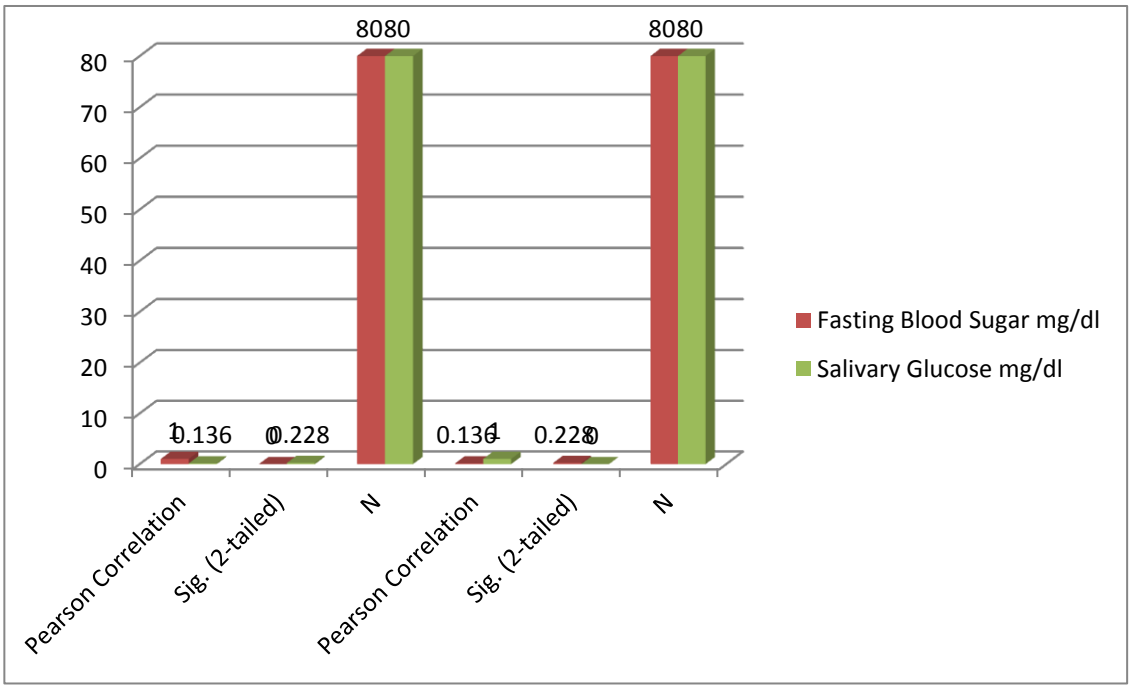
Graph 16: Correlation between Fasting Blood Glucose and Salivary Glucose in Group I (Healthy Controls)



Graph 17: Correlation between Fasting Blood Glucose and Salivary Glucose in Group II (Type 2 DiabetesMellitus)



Graph 18: Correlation between Salivary and Serum Glucose levels



Diabetes Mellitus is a complex group of syndromes that have in common, a disturbance in the body's use of glucose, resulting in elevated blood glucose levels. Blood glucose monitoring by the patient and the physician is an important aspect in the control of the devastating complications due to the disease. With ever improving advances in diagnostic pathology, the race for the next generation of bloodless, painless and accurate glucose instruments has begun. Most commonly used laboratory diagnostic procedures involve the analysis of blood, but other biological fluids are also being utilized for the diagnosis of other diseases and of these, saliva offers distinctive advantages.[74]

Knowledge of the components of saliva is important because they may indicate oral or systemic alterations, and it is also important because saliva may be a substitute for blood in lab tests for the diagnosis of illnesses. Several studies evaluate the concentration of glucose in the saliva of diabetic patients. Though differences in the output and composition of saliva from Diabetic and Non Diabetic subjects have been observed in a number of studies, many of these findings have been contradictory.[74]

Saliva offers some distinctive advantage as it can be collected non-invasively and by individuals with limited training However, studies pertaining to the use of saliva as a non invasive tool in monitoring blood glucose levels in diabetic patients have been done predominantly in the Western population.[9,10]

The aim of the present study was to compare the Salivary Glucose and Serum Glucose concentration in Non-Insulin dependent Diabetes Mellitus patients and the Non Diabetic Healthy individuals in a Chennai based population.

The study was conducted between March 2010 and April 2011 at Voluntary Health Services, Adyar, Chennai.

A case control study was conducted in which 80 patients were included. The study subjects were categorized into two groups out of which 40 were suffering from Type 2 Diabetes Mellitus and the other 40 were Non Diabetic Healthy individuals.

Participants with infectious diseases during one month before saliva sampling, active dental abscesses, and collagen vascular diseases were excluded from the study.

In India, the mean age of onset of Type 2 Diabetes in men is 44.0 ± 8.3yrs and in women the mean age is 42.9 ± 10.0yrs with a male predominance and a Male to Female ratio of 2:1. In our study, among the 80 subjects, 32 (40%) were males and 48 (60%) were females. The minimum age of the study subjects was 28yrs and the maximum age was 75yrs. The mean age among both men and women was found to be 50.97yrs, with a female predominance. This is in accordance with **Sreedevi et al**[71] whose patients had a mean age of 49.7yrs, though with a male predominance. The

gender variation could be attributed to the regional difference and the selection criteria in this study. **Cedric et al**[76] found a predominance of females in their study too with an age range of 56 ± 3 in males and 55 ± 2 in females. While **Michael W.J. Dodds**[79] saw a male predominance in their study though the mean age was similar to our study at 50.5 yrs. Similarly even **Veena et al**[74] found that the mean age of the diabetics was 51.65 ± 10.22 with a male predominance.

The current study involved subjects with Type 2 Diabetes Mellitus and a Healthy control group. The control group had a mean Fasting serum glucose level of 101.63mg/dl in males and 93.54mg/dl in females when compared to the Diabetic group who were found to have a mean Fasting serum glucose level of 208.13mg/dl in males and 197.67mg/dl in females which was significantly higher with a p value <.005. This is in accordance with **Hegde et al**[73] who found that the control group had a Fasting serum glucose level of 99.71 ± 6.92 while the Diabetic group had a value of 144.31 ± 53.55 with a significant p value <.005 when compared to the Healthy control group. **Sreedevi et al**[71] found that the Fasting serum glucose had a mean of 105.7 ± 22.3mg% in the control group while it was significantly higher with a value of 309.5 ± 68.2mg% in the Diabetics and a p value <.005. **Veena et al**[74] found that the Fasting serum glucose level among the control group was 95.58 ± 12.01mg/dl and that among the Diabetics ranged between 180-200mg/dl with a p value of <.005. **Nakamoto et al**[83] also

found the Fasting serum glucose level to be 115.7 ± 35.7mg/dl in the Diabetic group which was significantly higher than the control group with a p value of <.005.

Despite the significant differences in Fasting serum glucose levels, the Salivary glucose (SG) levels did not differ and were comparable between the two groups in our study. In this study, the average salivary glucose level found among the Healthy male and female individuals was 2mg/dl, while in the Diabetics it was found to be 2.5mg/dl in males and 2.2 mg/dl in females respectively with an insignificant p value >.005. While in a study done by **Maria-Sueli-Marques Soares et al**[84] to evaluate the salivary glucose level in unstimulated saliva of healthy individuals, the average Salivary Glucose level was found to be at a higher level compared to our study at 5.94mg/dl. An explanation for these differences may be the choice of certain study designs, as well as the diversity of the methods and criteria for selecting the samples. In this study, the measurements were recorded with the whole unstimulated flow rate. We have observed in our study that the modifications of the levels of capillary glycemia are not reflected in the saliva, which is in accordance with **Maria-Sueli-Marques Soares et al**[84]. On the other hand, **Sreedevi et al**[71] found the Salivary glucose level to range between 0.7 to 1.3mg% in the control group while in the Diabetic group it ranged between 1.5 to 8.0mg% with a significant p value <.005. **Sashikumar et al**[72] found the Salivary glucose levels to range between 1.5–25.6mg/dl in the Diabetic group while it was 0.2–7.7mg/dl in the

Control group with an insignificant p value >.005. The levels of Salivary glucose in the control group is similar to the one found in our study and this could be attributed to the fact that this study was also done in a chennai based population. **Veena et al**[74] found in their study that the Salivary glucose level ranged between 4.1 to 13.3mg/dl in the Control subjects while in the Diabetic group it was found to be 4.1 to 26.6mg/dl with a significant p value <.005. **Sathya Priya et al**[75] found that the Salivary glucose level ranged between 5.91–8.15mg/dl in the Control group while in the Diabetic group it ranged between 7.64-16.20mg/dl with a significant p value <.005.

With an increase in age, there was found to be a significant increase in the Fasting blood glucose levels in the control group, which is in accordance with **Arati S. Panchbai et al**[87] who found a significant p value <.005, signifying that with an increase in age there is a greater predisposition to diabetes.

An insignificant correlation with a p value >.005 was found between the Fasting blood glucose and the Salivary glucose levels in the Healthy control group. This is in accordance with **Hegde et al**[73] who found an insignificant p value >.005 and **Sashikumar et al**[72] who also found an insignificant p value >.005 in their study. But not in accordance with **Cedric et al**[76] who found a siginificant correlation with a p value <.005 between the Fasting blood glucose and Salivary glucose levels in their healthy group. **Veena v. Naik et al**[74] also in their study found a positive correlation between the Fasting blood glucose and the Salivary glucose with a p value

<.005. This variation in the study results could be attributed to the fact that this study unlike others included subjects from a Chennai based population who are known to have a carbohydrate rich dietary pattern.

Similarly an insignificant correlation was found between the Fasting blood glucose levels and the Salivary glucose levels in the Diabetic group with a p value >.005 which again is in accordance with **Hegde et al**[73] who also found no relation between the Fasting blood glucose and the Salivary glucose levels in their Diabetic subjects with a p value of >.005 but not in accordance with **Cedric et al**[76] who found a significant correlation between their Fasting blood glucose and Salivary glucose levels with a p value <.005. **Veena v. Naik et al**[74] in their study also had a highly significant p value < .005. Similarly **Aydin et al**[81] **a**lso found a significant correlation between the Fasting blood glucose and the Salivary glucose values with a p value <.005. **Sreedevi et al**[71]**, Nakamoto et al**[83]**, Amer et al**[80]**, Sashikumar et al**[72] **and Sathya Priya et al**[75] also found a significant correlation between the Fasting blood glucose and the Salivary glucose with a p value <.005.

In our study, an insignificant correlation was found between the Salivary glucose and the Fasting blood glucose levels in both Healthy and Diabetic group with a p value >.005. This is in accordance with **Hegde et al**[73] who concluded that the Diabetic group had significantly high Fasting serum glucose as compared to controls though despite these significant differences in Fasting serum glucose levels, the Salivary glucose levels did

not differ and were comparable between the two groups with an insignificant p value >.005. Our study is not in accordance with **Cedric et al**[76] who found that the glucose concentration in saliva is higher in Diabetic patients than in control subjects with a p value <.005. Neither is it, in accordance with **Arati S. Panchbhai**[87] who concluded that the Mean salivary glucose levels were clearly higher in Diabetics when compared to the Healthy Non Diabetics with a significant p value <.005. Similarly, **Veena v. Naik et al**[74] also concluded that a positive correlation was found between Blood glucose and Salivary glucose levels in both the Diabetics and the controls with a p value <.005 and that saliva could be used as an adjunct diagnostic tool in Diabetes Mellitus. **Sreedevi et al**[71] also concluded that as there was a significant correlation between Salivary glucose and Serum glucose with a highly significant p value <.005 and that Saliva does hold the potential of being a marker in diabetes. **Amer et al**[80] also found a significant p value of <.005 in their study and that the Salivary glucose concentrations seem to correlate with the Serum glucose concentration in the patients of Diabetes Mellitus. And finally, **Sashikumar et al**[72] found that Salivary glucose levels were significantly higher in Diabetics than Non Diabetics and that there was a significant positive correlation between Salivary and serum glucose levels with a p value <.005. The variation in the results could be attributed to a smaller sample size in our study as well as a variation in the study design, sample population in terms of female

predominance as well as regional variation due to a purely Chennai based population.

These findings confirm the poor link between glycaemia and glucose concentration or excretion in saliva, atleast on an individual basis. Nevertheless, the present study may well set the scene for further investigations on the regulation of glucose output from salivary glands, as well as on the potentially unfavorable effect of a high glucose salivary concentration on selected variables of oral health status in diabetic patients.

The present study titled "Comparison of Salivary Glucose and Serum Glucose concentration in Non-Insulin dependent Diabetes Mellitus patients" was conducted between March 2010 and April 2011 in the Out Patient Department of Voluntary Health Services, Adyar, Chennai to estimate the Salivary Glucose and Serum Glucose concentration in Non-Insulin dependent Diabeties Mellitus patients, to estimate the Salivary Glucose and Serum Glucose concentration in Healthy control group and finally to correlate these Salivary Glucose and Serum Glucose concentrations in Non-Insulin Dependent Diabetes Mellitus patients and healthy controls.

The study group comprised of a total number of 80 patients. Out of the 80 patients, 40 were Healthy controls and the other 40 were suffering from Type 2 Diabetes Mellitus. Informed consent was taken from all subjects before including them in the study. Participants with infectious diseases during one month before saliva sampling, active dental abcesses, and collagen vascular diseases were excluded from the study.

The experimental subjects were made to sit comfortably on a chair. Relevant demographic data was collected. An Intra Oral examination was carried out. Whole un-stimulated saliva was collected as well as the blood sample was collected. Saliva and blood samples were collected in sterile test tubes, immediately transferred aseptically to sterile tubes and frozen on dry ice and alcohol. The samples were stored in styroform boxes containing dry

ice and carried to a freezer where they are left until time of assessment of the Salivary glucose and Serum glucose.

The study documents the following data:

- Among the 80 subjects 48 (60%) were females and 32 (40%) were males.
- The minimum age among the subjects was 28yrs and maximum was 75yrs with a mean of 50.97 yrs.
- Among the 40 subjects in the Control group there was 1 male and 3 females in the below 35yrs category, 5 males and 4 females in the 36-45yrs category, 5 males and 8 females in the 46-55yrs category and finally 5 males and 9 females in the above 55yrs category.
- As this was an age and sex matched study, among 40 subjects in the Diabetic group there was 1 male and 3 females in the below 35yrs category, 5 males and 4 females in the 36-45yrs category, 5 males and 8 females in the 46-55yrs category and finally 5 males and 9 females in the above 55yrs category.
- The distribution of Salivary glucose among the 40 subjects in the Healthy controls ranged from 0mg/dl to 8mg/dl.
- Similarly, the Salivary glucose level distribution among the diabetics varied from 0mg/dl to 8mg/dl.

- The Mean fasting blood glucose among the healthy controls was 101.63mg/dl for males and 93.54mg/dl for females.
- The Mean fasting blood glucose among the diabetics was 208.13mg/dl for males and 197.67mg/dl for females.
- A significant correlation was found between Age and Fasting blood glucose among the Healthy controls.
- An insignificant correlation was found between the Salivary glucose and Fasting serum glucose level in the Healthy controls.
- An insignificant correlation was found between the Salivary glucose and Fasting serum glucose levels in the Diabetic group.
- Finally, an insignificant correlation was found between the Salivary glucose and Serum glucose.

To conclude, Salivary glucose concentrations showed no difference between the Type 2 Diabetic and the Control group. This implies that the association of high Fasting serum glucose with high Salivary Glucose levels is an infrequent observation which may be affected by metabolic control of the disease.

A large number of studies have been done in an attempt to check for the efficacy of Saliva as a diagnostic tool in Diabetes Mellitus. Unfortunately, there are no conclusive results. While the results of some studies give a go ahead to the usage of saliva as a diagnostic tool there are

others which disapprove of it. The need of the hour is larger studies that need to be performed in various parts of the world among different populations with different dietary patterns before a conclusive result is achieved.

Hence, the usage of salivary glucose as the only tool for evaluating glycemic status is debatable. Studies to compare long term indicators of glycemic status like HbA_1C, fructosamine levels with salivary glucose / glycated proteins should be under-taken with a larger sample size as well as keeping the importance of regional variations in mind.

1. Manfredi M, McCullough MJ, Vescovi P, Al-Kaarawi ZM and Porter SR. Update on Diabetes Mellitus and related Oral Diseases. Oral Dis 2004; 10:187-200.
2. Sacks DB and McDonald JM. The pathogenesis of Type II Diabetes Mellitus: a polygenic disease. Am J ClinPathol 1996; 105:149-56.
3. Betteridge DJ. Diabetes: Current perspectives. N Engl J Med 2000; 342:1533.
4. V. Mohan, S. Sandeep, R. Deepa, B. Shah and C. Varghese. Epidemiology of Type 2 Diabetes: Indian scenario. Indian J Med Res 125, March 2007, pg 217-230
5. Jukka H. Meurman, Hanna-Leena Collin, Leo Niskanen, Jari, Pekka Alakuijala, Sirpa Keinfinen and Matti Uusitupa. Saliva in Non-Insulin-dependent Diabetic patients and control subjects. The role of the autonomic nervous system. Oral Surg Oral Med Oral Pathol Oral RadiolEndod 1998;86:69-76.
6. Tencate R. Oral histology, Development, Structure and Function. 5th ed. Missouri: Mosby Publication, 1998. P. 315-44.
7. Kaulman E and Lamster IB. The Diagnostic Applications of Saliva. Crit Rev Oral Biol med 2002; 13: 197-212.
8. Van der Waal I. Diseases of Salivary Glands including Dry mouth and Sjogrens's Syndrome, Diagnosis and Treatment. New York: Springer; 1997. P. 23.

9. Darwazeh AMG, Macfarlane TW, McCuish A and Lamey PJ. Mixed Salivary Glucose levels and Candidal carriage in patients with Diabetes Mellitus. J Oral Pathol Med 1991;20:280-3.
10. Andersson A, Birkhed D, Berntorp K, Lindgarde F and Matsson L. Glucose concentration in parotid saliva after glucose/food intake in individuals with glucose intolerance and Diabetes Mellitus. Eur J Oral Sci 1998;106:931-7.
11. B.M. Frier and M. Fisher. Diabetes Mellitus. Davidson's Principles and Practice of Medicine. 20th Edition. Elsevier. 2006, 805-848.
12. Samuel J and McKenna. Dental management of patients with Diabetes.Dent Clin N Am 50 2006; 591-606.
13. The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Follow-up report on the diagnosis of Diabetes Mellitus. Diabetes Care 2003;26:3160–7.
14. American Diabetes Association. Standards of medical care in Diabetes. Diabetes Care 2006;29:S4–42.
15. American Diabetic Association. Screening for Type 2 Diabetes. Diabetes Care 2004;27:S11–4.
16. Rossini AA, Greiner DL and Friedman HP. Immunopathogenesis of Diabetes Mellitus. Diabetes Reviews 1993;1:43.
17. Libman IM, Pietropaolo M and Trucco M. Islet cell Autoimmunity in white and black children and adolescents with IDDM. Diabetes Care 1998;21:1824–7.

18. Martin S, Wolf-Eichbaum D and Duinkerken G. Development of Type 1 Diabetes despite severe hereditary B-cell deficiency. N Engl J Med 2001;345:1036–40.
19. Eisenbarth GS. Type 1 Diabetes Mellitus: a Chronic Autoimmune Disease.NEngl J Med 1986; 314:1360–8.
20. Littorin B, Sundkvist G and Hagopian W. Islet cell and glutamic acid decarboxylase antibodies present at diagnosis of Diabetes predict the need for insulin treatment. Diabetes Care 1999;22:409–12.
21. Riley WJ, Maclaren NK and Krischer J. A prospective study of the development of diabetes in relatives with Insulin-Dependent Diabetes. N Engl J Med 1990;323:1167–72.
22. Feutren G, Papoz L and Assan R. Cyclosporinincreases the rate and length of remissions in Insulin-Dependent Diabetes of recent onset: results of a multicenter double-blind trial. Lancet 1986;328:119–24.
23. Davies JL, KawaguchiY and Bennett ST.Agenome-wide search for Human Type1 Diabetes susceptibility genes. Nature 1994;371:130–6.
24. Atkinson MA and Maclaren NK. The pathogenesis of Insulin-Dependent Diabetes Mellitus. N Engl J Med 1994;331:1428–36.
25. Dahlquist GG, Patterson C and Soltesz G. Perinatal risk factors for childhood Type1 Diabetes in Europe. The EURODIAB Sub-study 2 Study Group. Diabetes Care 1999;22:1698–702.
26. Szopa TM, Titchener PA and Portwood ND. Diabetes mellitus due to virusesdsome recent developments. Diabetologia 1993;36:687–95.

27. Genuth SM and Federman DD. Diabetes Mellitus. Scientific American Medicine. New York: WebMD; 2004. p. 1–35.
28. Genuth SM. Plasma insulin and Glucose profiles in Normal, Obese, and Diabetic persons. Ann Intern Med 1973;79:812.
29. Kahn CR. Banting lecture: Insulin Action, Diabetogenesis, and the Cause of Type II Diabetes. Diabetes 1994;43:1066–84.
30. Boden G. Pathogenesis of Type 2 Diabetes: Insulin Resistance. EndocrinolMetabClin North Am 2001;30:801–15.
31. Cavaghn MK, Ehrmann DA and Polonsky KS. Interactions between Insulin Resistance and Insulin Secretion in the development of Glucose Intolerance. J Clin Invest 2000;106:329–33.
32. Groop LC, Bottazzo GF and Doniah D. Islet Cell Antibodies identify latent Type 1 Diabetics in patients aged 35–75 years at diagnosis. Diabetes 1986;35:237–41.
33. Bennett PH. Epidemiology of Diabetes Mellitus. New York: Elsevier; 1990. Edition 1.Pg. 363.
34. Mokdad AH, Ford ES and Bowman BA. Prevalence of Obesity, Diabetes, and Obesity related health risk factors, 2001. JAMA 2003;289:76–9.
35. Chan JM, Rimm EB and Colditz GA. Obesity, Fat distribution and Weight gain as risk factors for Clinical Diabetes in men. Diabetes Care 1994;17:961–9.

36. DeFronzo RA and Ferrannini E. Insulin resistance. A multifaceted syndrome responsible for NIDDM, Obesity, Hypertension, Dyslipidemia, and Atherosclerotic Cardiovascular Disease. Diabetes Care 1991;14:173–94.
37. Eckel RH, Grundy SM and Zimmet PZ. The Metabolic Syndrome. Lancet 2005;365:1415–28.
38. Grossi S. Treatment of Periodontal Disease and control of Diabetes: an assessment of the evidence and need for future research. Ann Periodontol 2001;6:138–45.
39. Tsai C, Hayes C and Taylor GW. Glycemic control of Type 2 Diabetes and severe Periodontal disease in the US adult population. Commun Dent Oral Epidemiol 2002;30:182–92.
40. Taylor GW, Burt BA and Becker MP. Non-insulin dependent Diabetes Mellitus and Alveolar Bone loss progression over 2 years. J Periodontol 1198;69:76–83.
41. Karjalainen KM and Knuuttila ML. The onset of Diabetes and Poor Metabolic control increases gingival bleeding in children and adolescents with Insulin-Dependent Diabetes Mellitus. J ClinPeriodontol 1196;23: 1060–7.
42. Iughetti L, Marino R and Bertolani MF. Oral health in children and adolescents with IDDM: a review. J PediatrEndocrinolMetab 1999;12: 603–10.
43. Iacopino AM. Periodontitis and Diabetes interrelationships: Role of Inflammation. Ann Periodontol 2001;6:125–37.

44. Kao CH, Tsai SC and Sun SS. Scintigraphic evidence of poor salivary function in Type 2 Diabetes. Diabetes Care 2001;24:952–3.
45. Guggenheimer J, Moore PA and Rossie K. Insulin-Dependent Diabetes Mellitus and Oral soft tissue pathologies, part II: Prevalence and Characteristics of Candida and Candidal lesions. Oral Surg Oral Med Oral Pathol Oral RadiolEndod 2000;89:570–6.
46. Clark CM and Lee DA. Prevention and Treatment of the Complications of Diabetes Mellitus. New Engl J Med 1995;332:1210–7.
47. Nathan DM. Long-term Complications of Diabetes Mellitus. N Engl J Med 1993;328: 1676–85.
48. Krolewski AS, Warram JH and Christlieb AR. The changing natural history of nephropathy in Type 1 Diabetes. Am J Med 1985;78:785–94.
49. Kannel WB and McGee DL. Diabetes and Cardiovascular disease: the Framingham Study. JAMA 1979;241:2035–8.
50. Stamler J, Vaccaro O and Neaton JD. Diabetes, other risk factors, and 12-yr cardiovascular mortality for men screened in the Multiple Risk Factor Intervention Trial. Diabetes Care.1993;16:434–44.
51. Haffner SM, Lehto S and Ronnemaa T. Mortality from Coronary Artery Disease in subjects with Type 2 Diabetes and in Nondiabetic subjects without prior Myocardial Infarction.Nengl J Med 1998;339:229–34.
52. Delamaire M, Maugendre D and Moreno M. Impaired leucocyte functions in Diabetic patients. Diabet Med 1997;14:29–34.

53. Joshi N, Caputo GM and Weitekamp MR. Infections in patients with Diabetes Mellitus. N Engl J Med 1999;341:1906–12.
54. Mahmoud AAF, Waren KS and Rodman HM. Effects of Diabetes Mellitus on Cellular Immunity. Surg Forum 1975;26:548–50.
55. Grossi S. Treatment of Periodontal Disease and Control of Diabetes: An Assessment of the Evidence and need for future research. Ann Periodontol 2001;6:138–45.
56. Tsai C, Hayes C and Taylor GW. Glycemic control of Type 2 Diabetes and Severe Periodontal disease in the US Adult population. Commun Dent Oral Epidemiol 2002;30:182–92.
57. Taylor GW, Burt BA and Becker MP. Non-Insulin dependent Diabetes Mellitus and Alveolar Bone Loss progression over 2 years. J Periodontol 1198;69:76–83.
58. Navazesh M. Methods for collecting Saliva. Ann NY AcadSci20:72-77.1993.
59. Young JA and Van Lennep EW. The process of Secretion of Organic products. In: The Morphology of Salivary Glands. London, UK: Academic Press, pp. 124-144.1978.
60. Turner RJ, Paulais M, Manganel M, Lee SI, Moran A and Melvin JE. Ion and Water transport mechanisms in Salivary Glands. Crit Rev Oral Biol Med 4:385-391.1993.

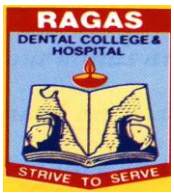
61. Zelles T, Purushotham KR, Macauley SP, Oxford GE and Humphreys-Beher MG. Saliva and Growth factors: the fountain of youth resides in us all. J Dent Res74:1826-1832.1995.
62. Baum BJ. Principles of Saliva Secretion. Ann NY AcadSci694:17-23.1993.
63. Drobitch RK and Svensson CK. Therapeutic Drug Monitoring in Saliva. An update. ClinPharmacokinet23:365-379.1992.
64. Marchetti P, Benzi L, Masoni A, Cecchetti P, Giannarelli R and Di Cianni G. Salivary Insulin concentrations in Type 2 (Non-Insulin-Dependent) Diabetic patients and Obese Nondiabetic subjects: Relationship to changes in Plasma Insulin levels after an Oral Glucose load. Diabetologia29:695-698.1986.
65. Marchetti P, Grossi C, Giannarelli R, Masoni A, Cristofani R and Giannecchini M. Salivary Immunoreactive Insulin: a new entry in Clinical Chemistry? ClinChem34:1478-1480.1988.
66. Fekete Z, Korec R, Feketeova E, Murty VL, Piotrowski J and Slomiany A. Salivary and Plasma Insulin levels in Man. BiochemMolBiolInt 30:623-629.1993.
67. Pasic J and Pickup JC. Salivary Insulin in Normal and Type I Diabetic subjects. Diabetes Care 11:489-494.1988.
68. Ellison PT. Measurements of Salivary Progesterone. Ann NY AcadSci694:161-176.1993.
69. FDI Working Group 10, Core. Saliva: its role in Health and Disease. Int Dent J 42:287-304.1992.

70. Chauncey HH. Salivary Enzymes. J Am Dent Assoc63:361- 369.1961.
71. Sreedevi, M.C. Shashikanth and P. Shambulingappa. Comparison of Serum Glucose and Salivary Glucose in Diabetic Patients. Journal of Indian Academy of Oral Medicine and Radiology. January – March 2008, Volume 20 Issue 1.
72. Sashikumar R and Kannan R. Salivary Glucose Levels are Unable to Predict Oral Candidiasis or Monitor Diabetes. Oral Surg Oral Med Oral Pathol Oral RadiolEndod 2010; 109(5):706-11.
73. Anupama Hegde, Ramya Shenoy, Prajwal D'Mello, Smitha A, Tintu A and Poornima Manjrekar. Alternative Markers of Glycemic Status in Diabetes Mellitus. Vol. 21, No. 3. 2010.
74. Naik Veena V. Satpathy Yasmin, Pilli Ganga and N. Mishra Mithilesh. Comparison and Correlation of Glucose levels in Serum and Saliva of patients with Diabetes Mellitus. Indian Journal of Public Health Research & Development Year:2011, Volume:2, Issue:1.
75. S.Sathyapriya, Dr.G.O.Bharani, M.Nagalingam, M.Jayanthi and Ms. U. Kanagavalli. Potential of Salivary Protein as a Biomarker in prognosis of Diabetes Mellitus. Journal of Pharmacy Research 2011,4(7),2228-2229.
76. Cedric Jurysta, Nurdan Bulur, Berrin Oguzhan, IlhanSatman, Temel M. Yilmaz, Willy J. Malaisse, and Abdullah Sener. Salivary Glucose Concentration and Excretion in Normal and Diabetic Subjects. Journal of Biomedicine and Biotechnology. Volume 67. 2009. Pg 123-125.

77. Campbell, M.J.A: Glucose in the Saliva of the Non-diabetic and the Diabetic Patient, Arch Oral Biol 10:197-205.1965.
78. A. M. G. Darwazeh, T. W. MacFarlane, A. McCuish, and P.J. Lamey, "Mixed Salivary Glucose levels and Candidal carriage in patients with Diabetes Mellitus," Journal of Oral Pathology & Medicine, Vol. 20, No. 6, pp. 280–283, 1991.
79. Michael W. J. Dodds and Anne P. Dodds. Effects of Glycemic Control on Saliva flow rates and Protein composition in Non-Insulin-Dependent Diabetes Mellitus. Oral Surg Oral Med Oral Pathol Oral RadiolEndod 1997;83:465-70.
80. S. Amer, M. Yousuf, P. Q. Siddqiui, and J. Alam, "Salivary Glucose concentrations in patients with Diabetes Mellitus— a minimally invasive technique for monitoring Blood Glucose levels," Pakistan Journal of Pharmaceutical Sciences, Vol. 14, No.1, pp. 33–37, 2001.
81. S. Aydin, "A comparison of Ghrelin, Glucose, Alpha-amylase and Protein levels in Saliva from Diabetics," Journal of Biochemistry and Molecular Biology, Vol. 40, No.1, pp. 29–35, 2007.
82. J. H. Meurman, H.L. Collin and L. Niskanen. "Saliva in Non-Insulin-Dependent Diabetic patients and Control subjects: The role of the Autonomic Nervous System," Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics, vol. 86, no. 1, pp. 69–76, 1998.
83. Ichiro Nakamoto, Kanehisa Morimoto, Tatsuya Takeshita and Masahiro Toda.Correlation between Saliva Glycated and Blood Glycated

Proteins.Environmental health and preventive medicine. Volume 8, Number 3, 95-9. 2005.

84. Maria-Sueli-Marques Soares, Mário-Márcio-Vasconcelos Batista-Filho, Marcele-Jardim Pimentel, Isabela- Albuquerque Passos and Eduardo Chimenos-Küstner.Determination of Salivary Glucose in Healthy Adults. Med Oral Patol Oral Cir Bucal. 2009 Oct 1; 14 (10):e 510-3.
85. C. M. Bennett, M. Guo and S. C. Dharmage. HbA_{1c} as a Screening Tool for detection of Type 2 Diabetes: a systematic review. Diabetic Medicine, 24, 333–34. 2007.
86. S. A. Mostafa, M. J. Davies, D. Webb, L. J. Gray, B. T. Srinivasan, J. Jarvis and K. Khunti. Original Article: Epidemiology The potential impact of using Glycatedhaemoglobin as the preferred diagnostic tool for detecting Type 2 Diabetes Mellitus. Diabet. Med. 27, 762–769 (2010).
87. Arati S. Panchbhai, Shirish S. Degwekar and Rahul R. Bhowte. Estimation of salivary glucose, salivary amylase, salivary total protein and salivary flow rate in diabetics. Journal of oral science, Vol 52, No. 3, 359-368, 2010.
88. Bloodcollection. http://www.ktl.fi/publications/ehrm/product2/part_iii4.htm.
89. Glucose (GOD – POD Method, End Point). KEE GAD Biogen Pvt. Ltd.



RAGAS DENTAL COLLEGE AND HOSPITAL

2/102 East Coast Road, Uthandi, Chennai – 600019

DEPARTMENT OF ORAL MEDICINE & RADIOLOGY

CASE SHEET PROFORMA

A. GENERAL INFORMATION

S.No: O.P.No: Date:

1. Name:
2. Age:
3. Sex: 1. Male : 2. Female:
4. Occupation:
    a. Unemployed
    b. Unskilled
    c. Skilled
    d. Professional
    e. Administration
    f. Trade/Business
    g. Student
5. Address:

6. Income;

    a. <Rs. 1,000/month b. >1,000-5,000/month

    c. >5,000/month

B. History:

1. History relating to Diabetes Mellitus

    a. Age at diagnosis of Diabetes Mellitus

    b. Onset and duration of Diabetes

    c. Family history of Diabetes Mellitus

2. Presence of any other systemic disease

    a. Present b. Absent

    If yes, specify

3. History of medication;

    a. Yes b. No

If yes, specify Duration Name of medicine

INVESTIGATION

1. Random Blood Sugar
2. Salivary glucose estimation
3. Fasting blood sugar
4. Post Prandial

CONSENT LETTER

I _____ the undersigned hereby give my consent for the performance of the diagnostic test on myself "to evaluate the **Serum and Salivary Glucose** test" conducted by Dr. Ruchi Gera under the able guidance of **Dr. S. Shanmugam**, M.D.S., HOD Department of Oral Medicine, Diagnosis and Radiology at the Ragas Dental College and Hospital, Chennai.

I have been informed and explained the status of my disorder, evaluation procedure, risk involved and likelihood of success. I also understand and accept this as a part of the study protocol, thereby voluntarily, unconditionally, freely give my consent without any pressure or fear in mentally sound and conscious state to participate in the study.

Witness/Representative **Patient's Signature**

(if any) **Date:**

ஒப்புதல் படிவம்

---------- என்கின்ற நான், சென்னை, ராகாஸ் பல்மருத்துவக் கல்லூரி மற்றும் மருத்துவமனையில் வாய் மருத்துவம் மற்றும் ஊடுகதிர் துறையின் பேராசிரியர் மரு. S. சண்முகம் அவர்களின் மேற்பார்வையில், முதுநிலை (M.D.S) பட்டப்படிப்பு பயிலும் திரு. ருச்சி கேரா அவர்கள் மேற்கொள்ளும், "**நீர்அழிவு நோயில் இரத்தம் மற்றும் உமிழ்நீரில் உள்ள சர்க்கரையின் அளவை கண்டறிதல்**" என்கின்ற ஆய்வுக்கு என் சம்மதத்தை தெரிவிக்கிறேன். மேலும், இந்த ஆய்வின், விளைவுகள் பற்றி அறிந்து இந்த பரிசோதனைக்கு நான் எந்தவித அச்சமுமின்றி தன்னிச்சையாகவும், தெளிவான முழு மனதுடன் என்னுடைய பரிபூரன சம்மதத்தினை அளிக்கிறேன்.

இப்படிக்கு

சாட்சியாளர்கள்

Date :

GROUP I

S. No	Name	Age	Sex	Fasting Blood Sugar mg/dl	Salivary Glucose mg/dl
1	Manomani	54	M	93	4
2	Neela	60	F	89	3
3	Suresh	35	M	89	1
4	Thondiammal	50	F	98	2
5	Devendran	47	M	83	1
6	Kamalavani	64	F	96	8
7	Mani	53	M	99	4
8	Shanti	48	F	100	7
9	Venkatraman	40	M	100	5
10	Vatsala	46	F	82	2
11	Adilakshmi	64	F	103	1
12	Srinivasan	48	M	107	2
13	Vardhraj	40	M	92	2
14	Lilly	60	F	97	1
15	Tamil Arasi	28	F	84	1
16	Thayar	40	F	98	3
17	C.Arjuna	56	M	124	0
18	Mangalakshmi	61	F	101	1
19	M. Shanti	49	F	99	0
20	Mahalingam	55	M	106	0
21	Kamala	60	F	90	2
22	Manavalan	65	M	107	3
23	Vadivel	65	M	119	1
24	P.Saroja	40	F	102	1
25	Selvanayagi	68	F	105	2

26	Jahida	40	F	107	1
27	Narayanaswamy	75	M	75	1
28	Mayappan	66	M	110	3
29	Anusuya	59	F	117	3
30	Sundari	54	F	86	2
31	S.Maheshwari	55	F	88	2
32	Deepa C.	30	F	75	1
33	Thondiammal C.	54	F	87	2
34	Manjula Bai	57	F	95	1
35	Madhusadan Rao	41	M	123	2
36	Maniya	50	F	95	1
37	Padma	40	F	81	1
38	Chinni	30	F	70	0
39	Srinivasan	43	M	107	1
40	Vardhraj	37	M	92	2

Group II

S. No	Name	Age	Sex	Fasting Blood Sugar mg/dl	Salivary Glucose mg/dl
1	Hemavathy	28	F	140	2
2	Perumal	65	M	146	2
3	P. Selvam	40	M	320	5
4	Shiva	35	M	220	2
5	J.Shanti	64	F	132	5
6	Jaya	60	F	240	2
7	Ranganayaki	50	F	195	2
8	Perumal	65	M	146	1
9	Selvi J.	40	F	240	3
10	Shanti R	61	F	169	2
11	Chellapan	48	M	171	2
12	Rajalakshmi	59	F	160	1
13	S. Duraipandi	37	M	310	1
14	Thirunangai	55	F	304	1
15	Jaya	46	F	139	1
16	V. Sundarrajan	43	M	218	2
17	Vallaiammal	64	F	163	2
18	Mallikam	54	F	300	2
19	Seetha	57	F	137	1
20	Sangavi	75	M	126	2
21	Usha	40	F	186	3
22	Arjunan	41	M	325	1
23	Masilamani	66	M	185	4
24	Nagammal	68	F	320	2
25	Baby	54	F	163	2

26	Paripuranam	60	F	280	4
27	Padmavathy	49	F	240	2
28	Nithyanandum	47	M	258	4
29	Sampath Kumar	48	F	212	3
30	Madhur	40	M	192	2
31	Shantanalakshmi	60	F	179	3
32	Manogi	40	F	150	2
33	Charuma	30	F	200	4
34	Saranya	50	F	150	2
35	Akshaya	30	F	156	2
36	Maneka	40	F	189	1
37	Manikandan	55	M	234	3
38	Hari	56	M	129	2
39	Thayagraj	54	M	200	5
40	Vimal	53	M	150	2